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MESENTERIC VASCULAR OCCLUSION

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Mesenteric vascular occlusion bears a very serious prognosis. Maingot[1] found 700 cases in the literature and, of those subjected to resections, only 8% survived. In 1938 Whittaker and Pemberton[2] described 19 cases of which 16 died.

In 1940 Thomas Moore,[3] in his masterly article on this condition wrote: 'The mortality resulting from this disease is still very high. This review is presented in the hope that further attention may be drawn to the condition. The present high mortality can only be reduced by earlier diagnosis and treatment'; yet, today the prognosis is not materially improved.

In the index of the Johannesburg General Hospital for the 6 years from 1953 to 1958, there are 24 cases listed under 'mesenteric thrombosis'; of these 24 cases 21 died.

HISTORY

Mesenteric vascular occlusion was first described by Tiedman in 1843 (quoted by Trotter[4]). In 1894 Elliott (quoted by Lower and Glazier[5]) described the first successful resection for this condition. In 1921 Klein[6] found only 24 cases of successful resection for the same condition. Since then a large number of cases has been reported. In 1940 Brown[7,8] found 722 cases in the literature.

INCIDENCE

Mesenteric vascular occlusion is not a rare disease; it is estimated to occur in 0·1% of all surgical admissions. This figure is, in all probability, an under-estimation, since many cases of transient abdominal pain, due to minor episodes of thrombosis, go undiagnosed.

The condition may be classified as follows:

Acute. (1) Arterial — due to embolism or thrombosis; (2) venous — due to thrombosis.

Chronic. Intestinal angina.

It is, therefore, quite incorrect to use the term mesenteric thrombosis when referring to this whole group of conditions. An effort should be made to use more precise nomenclature.

ANATOMY

Points of Interest

Mesenteric arterial occlusion may involve either the superior or the inferior mesenteric artery. Harkins[7] found that 1 in every 9 cases involved the inferior mesenteric artery. The greater incidence in the superior mesenteric artery is explained by the greater diameter of that vessel and the fact that it runs more nearly parallel to the aorta at its origin.

The superior mesenteric artery supplies the gut from the duodenum to the proximal two-thirds of the transverse colon. It may, however, be the only mesenteric artery and supply the whole of the small and large gut. Harkins reports a case in which this artery was occluded and the whole of the gut including the rectum was gangrenous.

The first branch of the superior mesenteric artery is the inferior pancreaticoduodenal artery which anastomoses with the superior pancreaticoduodenal artery to supply the duodenum. This branch may form a channel for the development of a collateral circulation when there is occlusion of the proximal part of the superior mesenteric artery. Several such cases are on record.

The venous drainage of the bowel is *via* the portal system. There are 2 significant points: (1) The portal system contains no valves, so that, if the pressure in the arterial system falls, due to arterial occlusion, blood flows back along the veins to cause congestion of the bowel; and (2) normally the pressure in the portal system is very low (*vide infra*).

PATHOLOGY

The effect of cutting off the flow of blood from an organ is dependent upon the rapidity with which a collateral circulation is established. A sudden occlusion is far more likely to produce an infarction than a gradually developing occlusion, because the collateral circulation may have time to develop before permanent damage occurs.

It may be thought that in the mesenteric vessels a small blockage could always be overcome, because of the extensive branching and anastomoses of the vessels. This is not so, since spreading secondary thrombosis usually occurs in these cases.

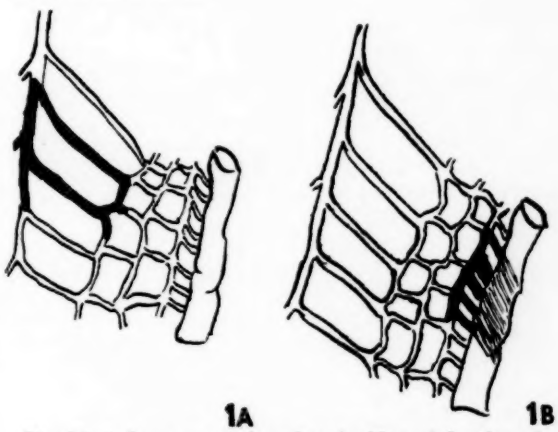

Fig. 1. A.* Large vessel thrombosed with no infarction of bowel. *B.* Vasa recti thrombosed with infarction of bowel.

* Adapted from Johnston and Baggenstos.[10]

There are 3 factors which favour the development of this spreading secondary thrombosis: (1) There is a fall in the pressure in the vessels following the block; (2) the pressure in the portal system is normally low; and (3) in the more extensive cases there is a loss of blood into the bowel causing oligaemic shock, with a further fall of blood pressure in these vessels.

If a number of vasa recti are blocked, the bowel supplied by them will become infarcted, but if some of the larger mesenteric vessels are occluded there may or may not be infarction of the bowel (Fig. 1).

There must indeed be many cases of this type which recover without operation or without being diagnosed.

Karcher[8] described a case which bears this out. Seven weeks after an attack of mesenteric vascular occlusion, the patient died of another cause. At autopsy there was a large thrombus in the superior mesenteric artery, but no infarction in the bowel.

This consideration suggests 2 interesting possibilities: (1) There may be sufficient blood supply to prevent infarction, but not enough to allow bowel function, thus causing a localized intestinal ileus; and (2) although the blood supply may be just adequate with a normal blood pressure, any fall in blood pressure at a later date may then cause an infarction of the bowel.

The infarction produced by mesenteric arterial occlusion is usually haemorrhagic. The bowel is infiltrated with blood, the mucosa is plum-coloured and swollen, and the lumen is filled with altered blood. Gradually areas of gangrene develop and infection spreads through the devitalized wall. There are similar changes in the mesentery if the obstruction is some distance from the gut. There is a blood-stained effusion into the peritoneal cavity.

The infarction may rarely be anaemic, and Trotter[4] reported 7 of these cases in the 359 cases he reviewed. In venous thrombosis the infarction is always haemorrhagic.

AETIOLOGY

The causes of mesenteric vascular occlusion may be summarized as follows:

A. Mesenteric Arterial Occlusion

1. *Embolism* may arise from: mitral valves, patent ductus, left auricular fibrillation, septic infarcts of lung, or atheromatous plaques on aorta.

2. *Thrombosis* is rare. It occurs as a result of: atherosclerosis, thrombo-angiitis obliterans, pressure by a tumour, diabetes, or dissecting aneurysm.

B. Mesenteric Venous Occlusion

1. *Primary*. 'There is no doubt that the number of primary cases will diminish the more complete and detailed is the search for the cause' — Moore.[3]

2. *Secondary*. Secondary cases may be the result of: portal obstruction, cirrhosis of the liver, polycythaemia vera, splenectomy, abdominal sepsis (such as acute appendicitis, ulcerative colitis, or strangulated bowel), or carcinoma of the rectum.

Gordin and Laurent[9] reviewed 47 cases of mesenteric vascular occlusion. Of 42 cases operated on, 37 were due to occlusion of the superior mesenteric artery and only 5 were due to occlusion of mesenteric veins.

In a review of 99 cases of mesenteric venous occlusion coming to autopsy, Johnston and Baggenstos[10] gave the aetiology as follows: infection, 28; neoplasms, 27; abdominal operations, 23; cirrhosis of the liver, 21; splenomegaly, 17; cardiac failure, 9; splenectomy, 5; and no cause found, 8. In some instances several probable causes were found in the same case.

AGE INCIDENCE AND MORTALITY

The age incidence is shown in Table I and the mortality rates in Table II.

TABLE I. AGE INCIDENCE OF CASES OF MESENTERIC VASCULAR OCCLUSION ACCORDING TO VARIOUS REPORTS

Report	*No. of cases*	*Ages*						
		Under 20	*20 - 30*	*30 - 40*	*40 - 50*	*50 - 60*	*60 - 70*	*70+*
Forty[19]	32	—	1	3	1	4	8	15
Johnston and Baggenstos[10] ..	99	5	10	8	19	32	19	6
Brown[20]	91	—	18	18	17	24	13	1
General Hospital, Jhbg. ..	24	—	1	—	6	4	7	6

TABLE II. MORTALITY OF CASES OF MESENTERIC VASCULAR OCCLUSION ACCORDING TO VARIOUS REPORTS

Date	*Report*	*No. of cases*	*Deaths*	*%*
1938	Whittaker[2] ..	19	16	84
1940	Moore[3]	8	4	50
1940	Brown[20]	104	?	68
1955	Maingot[1] ..	700	?	92
1957	Forty[19]	32	29	90
1959	Present series ..	24	21	87

CLINICAL FEATURES

Mesenteric vascular occlusion may present as follows: (1) symptomless — it may be discovered by aortography, (2) vague abdominal pain and nausea, (3) insidious onset with symptoms becoming worse until there is a fully developed infarction, (4) sudden abdominal catastrophe — this is the most typical type, or (5) intestinal angina.

The usual clinical presentation is an urgent abdominal catastrophe. There is sudden onset of persistent central abdominal pain, vomiting, and constipation or the passage of a little altered blood in the stool. There may be profound shock and sweating. The patient is pale and restless, with a low blood pressure, and a rapid pulse of poor volume. The abdomen becomes slightly distended, tenderness and rigidity are not marked, and bowel sounds are excessive. Later, signs of generalized peritonitis develop.

DIAGNOSIS

Trotter[4] reviewed 359 cases of mesenteric vascular occlusion. In only 4% was the diagnosis made preoperatively or before autopsy. Nevertheless, if the condition is kept in mind it should be recognizable.

Predisposing causes should be looked for, e.g. cardiac lesions or the history of loss of limbs or toes through vascular disease. The two-enema test may produce a little altered blood. The white-cell count is raised. X-ray of the abdomen may show an isolated dilated loop. Shaw and Rutledge[18] stated that there are few bowel shadows on X-ray.

TABLE III. CASES OF MESENTERIC VASCULAR OCCLUSION RECORDED IN THE INDEX OF THE JOHANNESBURG GENERAL HOSPITAL, 1953 - 58

Case	*Sex Age*	*Clinical picture*	*Diagnosed*	*Treatment*	*Result*
1	M 41	Acute appendicitis followed by generalized abdominal pain	At autopsy	IV drip and suction	Died on 8th day
2	M 43	Abdominal pain for 5 days. Vomiting	At laparotomy	44 in. of small bowel resected. Heparin	Died 24 hours postoperatively
3	M 46	Resection for mesenteric thrombosis 3 months before	Not confirmed	Symptomatic	Died 3 months postoperatively
4	M 66	Abdominal pain for 6 hours. Melaena	Not confirmed	Symptomatic	Died 2nd day
5	M 50	Abdominal pain for 3 days	Confirmed at autopsy	Symptomatic	Died 1st day
6	F 70	CCF, fibrillating. Abdominal pain	Not confirmed	Symptomatic	Died 1st day
7	M 44	CCF, mitral stenosis. Abdominal pain for 4 hours	Not confirmed	Symptomatic	Died 6th day
8	F 84	Abdominal pain for 3 days	Not confirmed	Symptomatic	Died 3rd day
9	F 66	CCF, aortic stenosis. Abdominal pain for 1 day	Not confirmed	Symptomatic	Died after 8 hours
10	M 44	Abdominal pain for 5 days	At laparotomy	18 in. of bowel resected. Heparin postoperatively	Died 6th postoperative day
11	M 87	Auricular fibrillation. Abdominal pain for 2 days	Not confirmed	Heparin	Died 2nd day
12	M 63	Abdominal pain for 2 days. Previous arteriosclerosis	At laparotomy	8 in. of bowel resected	Died 4 weeks postoperatively
13	F 80	Abdominal pain for 2 days	Not confirmed	Symptomatic	Died 2nd day
14	F 56	Mitral stenosis and fibrillation. Abdominal pain	At autopsy	Symptomatic	Died 10th day
15	F 24	Mitral stenosis. Sudden collapse. Melaena	Not confirmed	Symptomatic	Died in 6th week
16	F 71	Admitted comatose. Had complained of abdominal pain	Not confirmed	Symptomatic	Died after 1 hour
17	M 67	Coronary thrombosis. Arteriosclerosis. Abdominal catastrophe	Not confirmed	Had dicoumarol	Died in 2nd week
18	M 68	CCF. Melaena. Collapse	Not confirmed	Symptomatic	Died 1st day
19	M 65	Abdominal pain for 2 days. Vomiting	Confirmed at laparotomy	All except 18 in. of small bowel resected	Recovered
20	F 59	Cerebral thrombosis 2 days previously. Abdominal pain	Not confirmed	Symptomatic	Recovered
21	F 59	Rheumatic heart fibrillating. Abdominal pain. Melaena	Not confirmed	Heparin. Dicoumarol	Recovered
22	F 64	Abdominal pain for 2 days	At autopsy	Symptomatic	Died 2nd day
23	M 73	Abdominal pain for 7 hours. Melaena	Not confirmed	Symptomatic	Died 6th day
24	M 42	Obliterative vascular disease. Both legs previously amputated. Abdominal pain for 4 days	Pre-operatively	Resection of 4 ft. of small bowel	Died 2nd postoperative day

CCF = Congestive cardiac failure.

Abdominal Angina

In 1936 Dunphy and Whitfield[11] described 12 patients who had died of acute mesenteric occlusion. Seven of these patients had been seen, complaining of the following symptoms for a few months: recurrent abdominal pain after meals, weight loss, and occasional diarrhoea. Clinical examination and X-ray of the abdomen had shown nothing abnormal.

These patients had been suffering from abdominal or intestinal angina, due to ischaemia of the bowel. This is now a well-established clinical entity and an endeavour should be made to diagnose and treat this condition before the disaster of acute mesenteric vascular occlusion occurs.

Berman and Russo[12] recorded a case in which the diagnosis was made at laparotomy — no resection was done. The patient was alive and well 6 years later on anticoagulant therapy.

Endarterectomy is a practical procedure before the onset of an acute mesenteric vascular occlusion, whereas the mortality in emergency surgery may be greater than that of bowel resection.

TREATMENT

As may be expected in a condition with such poor prognosis, much has been written on the treatment of acute mesenteric occlusion and there is a great divergence of opinion.

Thomas Moore[3] recommends wide resection and anastomosis. Russell[13] says there is general agreement that if possible the bowel should be resected and anastomosed. Murray[14] recommends starting heparin therapy during the operation. Laufman and Scheinberg[15] make the point that heparin should only be given if bowel has been resected because there will be an increased blood loss into the bowel if the devitalized bowel remains. Aird[16] states that at Hammersmith Hospital, London, cases are treated on heparin, blood transfusion and antibiotics and only go to laparotomy if peritonitis develops. However, he admits that results of surgery are slightly better. Gordin and Laurent[9] state that heparin should be used postoperatively and, when there is massive infarction, heparin only should be used.

Mesenteric Embolectomy

Klass,[17] in 1951, was the first to undertake embolectomy in acute mesenteric vascular occlusion. His 2 patients died, 1 of ventricular failure and the other of haemorrhage from the mesenteric vein. Shaw and Rutledge[18] reported the first survival after mesenteric embolectomy in 1957. Since then they have had other survivals. Their cases were treated on anticoagulants given immediately after operation.

The success of embolectomy does not only depend on surgical technique but also, and primarily, on early diagnosis. The operation must, of course, be done before irreversible changes have occurred in the bowel. Immediate restoration to unquestionable viability is unlikely and so it is generally necessary to re-explore the patient's abdomen 24 hours later, to ascertain whether the bowel has survived.

CASES AT JOHANNESBURG GENERAL HOSPITAL 1953 - 1958

There were 24 cases of which 21 died. The diagnosis was confirmed by operation or autopsy in only 9 cases. However, in several of the others, there was very strong presumptive evidence of the correct diagnosis having been made (Table III).

Of the 9 confirmed cases 5 had bowel resections with 1 survival, while 4 cases treated conservatively came to autopsy.

It would be misleading to exclude the unconfirmed cases from this series as that would exclude the cases treated successfully by conservative measures and those cases for which permission for autopsy could not be obtained.

Comparing the cases at the Johannesburg General Hospital with those reported by Frank Forty,[19] we find that of 19 cases not operated on in Johannesburg, 17 died, while of 19 in Frank Forty's series who were not operated on, all died. Five cases in Johannesburg had bowel resection — of these 4 died; in Frank Forty's series, 6 cases had bowel resection and of these 4 died. Frank Forty reports a laparotomy only in 7 cases; of these 6 died.

At the Johannesburg General Hospital heparin was given to only 5 cases; of these 4 died and 1 survived.

Of the 5 cases subjected to operation, only 2 were diagnosed pre-operatively, and 1 of these 2 survived. Many cases were unsuitable for surgery but in several the diagnosis was missed until it was too late.

These cases of mesenteric vascular occlusion at the Johannesburg General Hospital present the same gloomy picture as those described elsewhere. The delay in diagnosis is partly responsible for the very poor prognosis and irrespective of other advances this is the main factor which must be improved before we can expect any fall in the high mortality.

SUMMARY

1. A brief review of the literature is given, with a review of the aetiology, clinical features, diagnosis and treatment of mesenteric vascular occlusion.

2. A plea is made for the use of more precise nomenclature in referring to this condition.

3. There were 24 cases of mesenteric vascular occlusion at the Johannesburg General Hospital between 1953 and 1958, of which 21 died. The delay in diagnosis is partly responsible for the very poor prognosis.

I wish to thank Prof. D. J. du Plessis and Mr. D. Tanne for their helpful criticism and advice. My thanks are also due to Dr. K. Mills, Superintendent of the Johannesburg General Hospital, for permission to review these cases.

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VAN DIE REDAKSIE : EDITORIAL

TERUGGEHOUE MISKRAAM

Die toestand van die dood van die embrio sonder uitdrywing van die produkte van bevrugting binne 'n redelike tyd, wat bekend staan as teruggehoue miskraam, is nooit baie duidelik omskryf nie. Daar word egter gewoonlik aangeneem dat dit ses weke lank duur. Dit kan ook maande lank duur. Die feit van die saak is dat dit klinies dikwels moeilik is om seker te wees van die diagnose voordat ses weke verloop het. Die toestand gaan oor in 'n voorgeboortelike fetale dood gedurende die laaste helfte van swangerskap.

Wanneer die dood van die embrio of fetus die eerste verskynsel is by 'n proses van miskraam, volg terughouding gewoonlik vir 'n tydperk wat varieer van ongeveer twee weke tot ses weke.

Tot ongeveer die derde of vierde maand van swangerskap kan dit baie moeilik wees om 'n differensiële diagnose tussen 'n normale swangerskap aan die een kant, en dreigende miskraam, onvolledige miskraam, buitebaarmoederlike swangerskap, trosvormige mola, skynswangerskap, of teruggehoue miskraam, aan die ander kant, te maak. Dit is veral waar as 'n mens die pasiënt nog nie vantevore ondersoek het nie, en veral as die pasiënt naby die menopouse is. Die grootte van die uterus moet akkuraat by elke ondersoek vasgestel word en die gebrek aan die normale uitsetting van die uterus moet verdag wees. Definitiewe inkrimping van die grootte van die uterus oor 'n tydperk is 'n baie definitiewe diagnostiese bevinding.

Die dokter behoort besonder versigtig te wees om nie 'n oorhaastige diagnose van teruggehoue miskraam te maak nie aangesien selfs die afwesigheid of ophou van die subjektiewe tekens van swangerskap, soos naarheid, of veranderinge in die borste, of fetale bewegings, veral in die middelste maande, nie noodwendig fetale sterfte aandui nie. Die definitiewe afwesigheid van fetale hartklanke, as hulle sonder twyfel voorheen gehoor is, is sterk bykomstige bewys van fetale dood. Verdere ondersoeke bestaan uit bepaling van chorioniese gonadotrofien en röntgenondersoeke. Bepalings van chorioniese gonadotrofien word gewoonlik negatief binne 'n week nadat die ovum dood is, of dit nou ook al uitgedryf of teruggehou is; somtyds bly 'n swak positiewe reaksie selfs maande lank voortbestaan in die teenwoordigheid van teruggehoue miskraam. Hierdie reaksie dui die bestaan van aktiewe chorioniese weefsel aan en nie noodwendig 'n lewensvatbare ovum of fetus nie. 'n Swak positiewe of 'n negatiewe reaksie mag ook by 'n normale swangerskap voorkom. Die urinetoets is dus nie altyd 'n betroubare diagnostiese hulpmiddel nie. Sommige röntgenologiese waarnemings is egter dikwels waardevol, bv. oorvleueling van die fetale skedelbene (Spalding se teken); hierdie teken is egter gewoonlik betroubaar net in die laaste weke van swangerskap. 'n Beter teken is kollaps van die fetale geraamte met 'n abnormale houding van fleksie en somtyds die teenwoordigheid van gas in die fetale buik. Knettering van die skedelbene lewer afdoende bewys. In die meeste gevalle egter kan 'n diagnose eers gemaak word nadat die grootte van die uterus by meer as een geleentheid na geskikte tydperke bepaal is.

Terughouding van die dooie fetus veroorsaak, as dit ongesteurd gelaat word — anders as wat gewoonlik geglo word — geen skade nie. Die meeste gevalle sal spontaan eindig met baie min moeite, maar spontane lediging mag soms eers na verskeie maande voorkom. Sulke afwagtende behandeling mag knellend wees vir die moeder en familie, wat teen die gevare van aktiewe chirurgiese tussenkoms gewaarsku moet word, bv. bloeding en infeksie.

Die gebrek aan sametrekking van die uterus, wat oorspronklik verantwoordelik is vir die toestand van teruggehoue miskraam, veroorsaak waarskynlik dat die uterus in atoniese toestand bly na gedwonge lediging, met die gevolg dat bloeding en daaropvolgende infeksie ontstaan.

Fetale weefsel sonder lewensvatbaarheid lei ook tot infeksie — die gasvormende bakterië is belangrik in hierdie verband. Dit is algemeen bekend dat die uterus in hierdie gevalle weerstandig is vir die oksitotiese middels.

Hipofibrinogenemie by die moeder, wat selde voorkom, met die gevolglike neiging tot bloeding, ontstaan ongeveer vier weke na die dood van die fetus teen die einde van die middelste trimester van swangerskap. Die moeder mag ook 'n sterk begeerte hê om so 'n swangerskap te beëindig. Die uterus mag sensitief wees vir hoë dosisse van ‚pitosien' wat met binneaarse indruppeling gegee word; of 'n mediese induksie met stilbestrol, of (aangesien die fetus alreeds dood is) met kina, kan probeer word.

Chirurgiese induksie word nie aanbeveel nie aangesien die besondere gevaar van infeksie, wat alreeds genoem is, bestaan. Met die ontwikkeling van abnormale bloedingsneigings, wat ontdek kan word deur toetse vir standaard bloedings- en stollingstye, moet konserwatiewe behandeling gestaak word. Binneaarse toediening van oksitotiese middels mag genoeg wees om spontane kraam en uitdrywing van die fetus te veroorsaak. Pogings om in sulke gevalle in te meng, moet nie aangedurf word sonder dat vars bloed of fibrinogeen beskikbaar is nie, aangesien die toestand, alhoewel dit selde voorkom, ernstig is. As dit om redes van geestespanning of om ander goeie redes nodig mag blyk om die uterus te ledig, moet chirurgiese lediging onder ideale toestande (en as dit tegnies moontlik is) onderneem word. In ander gevalle mag vaginale histerotomie nodig wees; abdominale histerektomie is selde te regverdig vir hierdie toestand.

THE NEWLY QUALIFIED DOCTOR AND THE MEDICAL ASSOCIATION

It has become a tradition for professional men all over the world to organize themselves into learned societies to safeguard their material interests and to provide a medium through which they can give expression to their cultural and scientific aspirations. In most of the countries of the Western world doctors have organized national

medical associations which are, in turn, members of the World Medical Association. In the same way we, in this country, have established the Medical Association of South Africa 'to promote the medical and allied sciences and to maintain the honour and interests of the medical profession'.

Since the early days of its existence it has been the explicit aim of the Medical Association to function as a responsible body of professional men who are fully aware of the great and important obligation which rests on them — to keep abreast of the times in scientific and cultural matters.

Admittedly, the Association has, in recent years, been subjected to severe scrutiny and criticism. It must, however, be borne in mind that it has had to face extremely difficult problems especially in the field of the economics of medical practice. The Association can only continue to deal with these problems on a satisfactory level if it can be assured of the whole-hearted support, not only of all its members, but also of each individual practising doctor.

The advantages of membership of the Medical Association have been well known to a large number of its members all over the country, but there are still many doctors who are unaware of these advantages. Furthermore, by the time this article is published, a large number of newly-qualified doctors will have joined the ranks of the medical practitioners of the country. It is to these two groups of doctors — those who qualified some time ago but who have not yet joined the Association, and those who have qualified recently — that we should like to extend a special invitation to become members of the Association.

In particular, we should like to draw the attention of all newly-qualified doctors to the excellent article on 'The Medical Association: its rôle in the past and its ideals for the future' which was published in the issue of the *Journal* for 21 May 1960 (**34**, 423). This article was written by Dr. J. H. Struthers, Past-Chairman of the Federal Council, and deals with the services rendered by the Association to the profession in the fields of the economics of medical practice, the publication of the *Journal*, the rôle of the Association in promoting medical education in the widest sense of the word, international affiliation, and the Association's hopes for the future.

Following is a brief summary of all the services which are at present being provided by the Association:

1. Opportunities for meeting colleagues, holding scientific meetings and providing a forum for the exchange of opinions.
2. A *Journal* for the spreading of medical knowledge.
3. Means for the settlement of ethical disputes between members.
4. Means for negotiating with medical aid societies and provision of some measure of control for medical benefit societies.
5. Means for negotiating with the Workmen's Compensation Commissioner.
6. Acting as the voice of the profession in all matters concerning medical practitioners, and being recognized as the official body in various Acts and Ordinances.
7. Legal protection for individual practitioners.
8. Procuring of income tax concessions of various kinds.
9. Obtaining preferential insurance of various forms for members.
10. Assistance to members by the Agency departments.
11. Amenities for members travelling overseas by reciprocity with the British Medical Association and the Canadian Medical Association, and through membership of the World Medical Association.
12. Improvement of salary scales of full-time personnel.
13. Influence on medical schools and medical education generally, e.g. encouraging and working towards the establishment of the College of Physicians, Surgeons and Gynaecologists of South Africa.
14. Postgraduate courses, provided directly or through medical schools.
15. Library facilities through grants to medical school libraries.
16. Assistance to needy dependants of members, through the Benevolent Fund.
17. Acting as a unifying factor, through Branches and Divisions, among practitioners.
18. Liaison with other professional bodies and the public.

The Association can succeed in playing a satisfactory and worth-while rôle in medical professional life only if it has the wholehearted support of *all the doctors* in the country.

OBSERVATIONS ON THE NUTRITIONAL STATE OF LOW-PAID AFRICAN LABOURERS IN NATAL

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In South Africa, as elsewhere, the problem of the nutrition and health of workers is at present arousing much interest. 'The modern approach to the problems of industrial health more and more tends to involve positive programs of health promotion ... There are thousands of employees who are operating with low efficiency as a result of ... inadequate dietaries'.[1]

This report deals with a study of the nutritional state and diet of 6 groups of low-paid African male labourers in or near Durban. The investigation was conducted during May 1960 at the request of 6 companies concerned about the diet, health, and efficiency of their workers. These companies were: company A, a whaling industry; B, a sugar-planting and milling company; C, a factory handling bulk raw materials; D, a foundry and engineering factory; E, a food factory; and F, a manufacturing industry.

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SUBJECTS AND METHODS

At each firm 40 labourers were selected for study at random from those in the 20 - 39 years age group. The selected men were on the lowest grade of pay, and employed for at least 6 months (4 months at company B). A few workers with occupational and possible dietary privileges, e.g. cooks, and also men under treatment for tuberculosis, were excluded.

The mean age of the men studied was 28·6 years. In each group 80% or more of the men were Zulu, except at company B where 68% were Pondo. Over half (62%) of the men had had no schooling, and only 18% had had over 4 years at school. Almost all (97%) of the men had spent the greatest part of their childhood years in a rural area. In each group, between 62 - 70% of the men were married. They were essentially migrant labourers. Only 6% of the married men, and 1% of the unmarried, were living with their families. Many of the men had spent long periods in town, except those at company B, of whom 98% had never lived in a town. Of those at the other companies, 90% had spent at least 5 years in a town, and 56% at least 10 years. Except at company B, where most workers are employed on contract for periods of 6 months, most of the men selected for study were in fairly stable employment; at the other firms they had been in their present job, on an average, for 4·6 years (median figure) exclusive of breaks. All the men were engaged in manual work, which was possibly least strenuous at companies E and F. The men's mean cash wages, according to their own statements, were £2 12s. per week, ranging from £1 1s. to £3 13s. at different companies.

Each man was questioned by a health educator trained in interview technique, and was subjected to a clinical nutritional assessment. Detailed observations were confined to selected signs which are frequently found in malnourished groups. These signs, together with explanatory notes, are listed in Table I. Fuller details of the methods used will be found elsewhere.[2] Scores were used to express the grade of 3 skin abnormalities, viz. phrynoderma, dyssebacea/folliculosis and skin xerosis. These scores were arrived at by recording the intensity of each sign (1 = mild, 2 = moderate, and 3 = marked) in a number of selected skin areas,[2] and then adding the corresponding figures for the various skin signs. The total was used as a measure of the grade of each abnormality. The 'skin xerosis' score was based on the total scores for the following signs: dryness, dullness, increased reticulation, flaking and hyperkeratosis. This score was thus an index of skin dryness and dullness, and the commonly associated textural abnormalities. Each man was examined simultaneously by both the authors, who reached agreement on each finding. Blood was collected for estimations of serum protein (Weichselbaum's method[3]); unfortunately, fractionations were not possible.

The chi-square test, with Yates' correction, was used to assess the statistical significance of the findings. The 5% level of probability was used.

FINDINGS

Signs of malnutrition were found in all the men examined. There was a high prevalence of leanness, of skin and mucosal lesions, and of low total serum-protein concentrations (Table I). No cases of pellagra or other flagrant nutritional diseases were found. Few such cases, however, are to be expected in a group of active employed men.

The findings were compared with those among a population sample of African men living in a Durban housing scheme, studied in 1958. Most (84%) of the men in this sample were in social classes IV or V[11] (predominantly unskilled or semi-skilled labourers). The findings in the 2 groups were in many respects similar. However, leanness, gingivitis and dyssebacea/folliculosis were more common than in the population sample, and follicular enlargement and fissuring of the tongue less common (Table I).

The men's responses to a series of questions concerning the frequency with which they usually took a number of selected foodstuffs indicated that their diet was, in general, unsatisfactory, with particular regard to animal tissues, milk, yellow or green vegetables, and fruit. The average number of days a week on which the following foods were taken (median figures) were: meat, 3; fish and eggs, 0; milk as a beverage or with porridge, 0; *amasi* (sour milk), 0; green or yellow vegetables, 2; fruit, 0; brown or wholewheat bread, 3; and white bread, 0.

The prevalence of some signs of malnutrition showed a wide variation at the 6 companies studied.

Company A

Although there was evidence of malnutrition in this group, the prevalence of the various signs tended to be lower than in the other groups, significantly so in many instances (Table I).

These men were housed in a compound, and fully fed by the company. An analysis of their rations in March 1960 had revealed a number of shortcomings, which the company had attempted to rectify during April 1960 (the month preceding the present study), mainly by adding brown bread and *tshwala* (kaffir beer), and by increasing the allotment of meat and beans (Table II). In addition, pending further improvement in the rations, a daily vitamin pill was provided. The rations at the time of the present study were still deficient in calcium. The figures presented in Table II are an underestimate of the true value of the men's diet, since the men are liberally supplied with whale-meat (amount unknown), over and above their rations, during the whaling season (approximately May - September). As most of the men studied had been employed for some years (90% for more than 1 year, and 62% for more than 2), it was considered that their relatively good nutritional state could be ascribed partly to the cumulative effect of their liberal provision of whale-meat, and partly to the recent improvements in their general diet.

Company B

The men in this group, who were also housed in a compound and provided with their full diet, were in a worse nutritional state than those at company A. There was among them a significantly greater prevalence of low relative weights, angular stomatitis, skin xerosis, phrynoderma, dyssebacea/folliculosis, and low total serum-protein concentrations. Tongue lesions and pyorrhoea were less prevalent than among men at companies C - F.

An analysis of the rations provided at this plantation in 1958 had revealed gross shortcomings in the diet (Table II). A high state of malnutrition had been found in a

TABLE I. PREVALENCE % OF SIGNS OF MALNUTRITION AMONG LABOURERS, WITH COMPARATIVE FIGURES FOR RESIDENTS IN AN URBAN HOUSING SCHEME

	Labourers (combined)	*Durban housing scheme**	*Company A*	*Company B*	*Company C***	*Company D*	*Company E*	*Company F*	*Explanatory notes*
Number of labourers ..	240	65	40	40	40	40	40	40	
Leanness	30·9	7·7†	22·5	42·5	40·0	25·0	22·5	32·5	Arm skinfold thickness of under 5·0 mm. Measured half-way down the back of the right arm, over the triceps, with a Harpenden caliper.[4]
Underweight	17·5	12·3	*0*^{BCDEF}	**22·5**^{A}	**20·0**^{A}	**15·0**^{A}	**15·0**^{A}	**32·5**^{A}	Weight at least 10% below the standard weight of a US male of the same age and height.[5]
Overweight	17·1	24·6	25·0	12·5	30·0	12·5	10·0	12·5	Weight at least 10% above the standard weight of a US male of the same age and height.[5]
Lip signs									
Lip dryness	61·3	58·5	*42·5*^{C}	67·5	**82·5**^{ADEF}	*62·5*^{C}	*52·5*^{C}	*60·0*^{C}	Any degree of dryness of the whole of the lips or at angles.
Angular stomatitis ..	9·2	8·3	*0*^{B}	**22·5**^{AC}	*0*^{B}	10·0	10·0	12·5	Sodden or cracked angles. Over 5% is regarded as abnormal.[5]
Gum signs									
Gingivitis	63·3	40·0†	65·0	65·0	77·5	55·0	65·0	55·0	Any degree of softness and swelling or redness, with or without pus.
Pyorrhoea	46·2	52·3	*25·0*^{CDE}	*27·5*^{CBE}	**70·0**^{ABF}	55·0^{AB}	55·0^{AB}	*45·0*^{C}	Pus obvious, or appearing when gums are pressed lightly.
Bleeding	7·1	6·2	7·5	**17·5**^{EF}	2·5	**15·0**^{EF}	*0*^{BD}	*0*^{BD}	Bleeding, spontaneously or on light pressure.
Tongue signs									
Papillary changes ..	81·2	—	*65·0*^{CDEF}	*65·0*^{CDEF}	**90·0**^{AB}	**87·5**^{AB}	**92·5**^{AB}	**87·5**^{AB}	Papillary atrophy and/or hypertrophy/hyperaemia, of any degree. Usually at the tip and sides. 'Geographism' excluded. As atrophy and hypertrophy/hyperaemia may under certain conditions be manifestations of the same pathology, their combined prevalence is given, as well as the separate prevalence of each (see below).
Ditto (moderate or marked)	12·5	—	7·5	*2·5*^{CD}	**20·0**^{B}	**20·0**^{B}	15·0	10·0	Excluding papillary atrophy at the tip and sides only, or slight hypertrophy or hyperaemia.
Papillary atrophy ..	55·0	—	*35·0*^{CE}	*47·5*^{C}	**77·5**^{ABD}	*50·0*^{C}	65·0^{A}	57·5	Of any degree. Usually at the tip and sides. 'Geographism' excluded.
Ditto (moderate or marked)	3·8	—	0	2·5	2·5	5·0	10·0	2·5	Excluding signs at the tip and sides only.
Papillary hypertrophy or hyperaemia ..	58·8	—	*52·5*^{D}	*37·5*^{DF}	*52·5*^{D}	**77·5**^{ABC}	62·5	**70·0**^{B}	Of any degree.
Fissuring	66·2	80·0†	62·5	60·0	*50·0*^{DE}	**80·0**^{C}	**77·5**^{C}	67·5	Of any degree.
Magenta	6·2	0	5·0	*0*^{C}	**15·0**^{B}	2·5	7·5	10·0	Over 0% is regarded as abnormal.
Indentations	92·5	100·0	100·0	97·5	87·5	100·0	95·0	100·0	Tooth impressions visible at sides or tip.
Skin signs									

Papillary hypertrophy or hyperaemia	58·8	—	*52·5*[D]	*37·5*[DF]	*52·5*[D]	**77·5**[ABC]	62·5	**70·0**[B]	Of any degree.
Fissuring	66·2	80·0†	62·5	60·0	*50·0*[DE]	**80·0**[C]	**77·5**[C]	67·5	Of any degree.
Magenta	6·2	0	5·0	*0*[C]	**15·0**[B]	2·5	7·5	10·0	Over 0% is regarded as abnormal.
Indentations	92·5	100·0	100·0	97·5	87·5	100·0	95·0	100·0	Tooth impressions visible at sides or tip.
Skin signs									
Xerosis	79·2	81·3	*35·0*[BCDEF]	**85·0**[A]	**87·5**[A]	**90·0**[A]	**90·0**[A]	**87·5**[A]	Score based on degree and extent of dryness, dullness, increased reticulation and flaking on face, trunk, shoulders and arms, and thighs.[2] Men with scores of under 9 (i.e. mild or localized signs) are excluded.
Xerosis (marked) (thighs)	17·9	—	*0*[BCEF]	**22·5**[A]	**35·0**[AD]	*10·0*[C]	**15·0**[A]	**25·0**[A]	Marked signs on thighs (score of 8 or more for this skin area). Separate figures provided for thighs, which are relatively protected from occupational trauma.
Phrynoderma	38·3	32·8	*7·5*[BDEF]	**72·5**[ACDF]	*17·5*[BEF]	35·0[AB]	**57·5**[AC]	40·0[ABC]	Score based on degree and extent of follicular keratosis[6] on trunk, shoulders and arms and thighs, excluding elbow, knee and trochanteric regions, and sternal and interscapular areas. Score based on degree and extent of lesion.[2] Men with scores of under 2 (i.e. mild localized signs) are excluded.
Ditto (moderate or marked) (thighs)	28·8	—	*7·5*[BE]	**65·0**[ACDEF]	*17·5*[B]	*20·0*[B]	37·5[AB]	*25·0*[B]	Men with scores of 2 or more for this skin area. Excluding knee and trochanteric regions.
Follicular enlargement	27·9	50·0†	25·0	25·0	*17·5*[E]	25·0	**45·0**[C]	30·0	'Permanent gooseflesh'[6] of any degree in areas listed above, under 'phrynoderma'.
Dyssebacea/folliculosis (moderate or marked)	41·7	26·6†	**42·5**[C]	**60·0**[CF]	*12·5*[ABDEF]	**47·5**[C]	**52·5**[C]	35·0[BC]	Follicles enlarged or plugged with sebum. on face, sternal or interscapular areas. Score based on degree and extent of lesion.[2] Men with scores of under 4 are excluded.
Mosaic (legs)	71·2	—	57·5	60·0	75·0	75·0	80·0	80·0	'Crackled skin'.[6] Mild lesions are excluded.
Oedema	1·7	3·1	2·5	2·5	0	2·5	2·5	0	Definite pitting on pressure over both ankles. Over 0% is regarded as abnormal.[5]
Low total serum proteins (below 7·5 g.%)	39·2 (227)	—	*5·1*[BDEF] (39)	23·7[ADF] (38)	*22·5*[DF] (40)	60·5[ABCEF] (38)	32·4[ADF] (37)	**97·1**[ABCDE] (35)	Although there is no clarity on the precise significance of total serum-protein values, there is evidence of a relationship between malnutrition and low values among Durban Africans.[7,8] The figures in parenthesis indicate the number of subjects.

Where there are significant differences in the prevalence of a sign at different companies, the significantly higher figures are printed in bold type, and the significantly lower figures in italics. Significant differences are indicated by superior letters, e.g. A= Significantly different from the figure for company A, B=Significantly different from the figure for company B.

* Figures for men aged 20 - 39, derived from a study of a population sample of adults living in a Durban housing scheme.[2,9,10]

** Many of the men were covered with dust which was difficult to remove completely, and obscured the skin signs. The figures for skin xerosis may be unduly high, and those for follicular lesions unduly low.

† Significantly different from the figure for the combined group of labourers.

TABLE II. ANALYSIS OF DAILY RATIONS OF MEN AT COMPANIES A AND B

	Company A		Company B		Recommended standards†	
	Before April 1960	*From mid-April 1960**	*February 1958*[12]	*April 1960***	*Moderately active*	*Heavy work*
Calories	1,937	3,887	4,876	6,081	3,000	4,500
Calories derived from maize (% of total calories)	62·7	58·4	76	70		
Protein (g.)	62·6	124	142·6	166·8	65	65
Animal protein (g.)	14·4	26·7	24·2	24·5	22	22
Calcium (mg.)	232	391	400	429	700	700
Iron (mg.)	19·7	34·5	43·8	53·3	9	9
Thiamine (mg.)	1·3	2·8	1·7	5·1	1·0	1·6
Riboflavin (mg.)	0·6	1·3	1·2	2·3	1·6	1·6
Nicotinic acid (mg.)	11·1	16·8	20·6	25·7	15	18
Ascorbic acid (mg.)	33·0	34·9	9·4	34·2	40	40
Vitamin A (i.u.)	647	649	74·6	1,330	4,000	4,000

Calculations based on Fox and Golberg's tables.[13] Where foods were not listed in these tables, other sources were used.[14-16]

* In addition to these rations, each man is supplied daily with a vitamin pill containing 2 mg. of thiamine, 3 mg. of riboflavin, 20 mg. of nicotinic acid, 75 mg. of ascorbic acid, 5,000 i.u. of vitamin A, and 400 i.u. of vitamin D.

** In view of the high caloric value, it is unlikely that most workers consume all of their rations. It is probable, however, that there is relatively little wastage of the more nutritious ingredients such as meat, fish, pulse and other vegetables.

† Recommended minimum daily dietary standards for men of average weight 160 lb.[17] These recommendations do not allow for vitamin losses during cooking; such losses may be considerable if food is overcooked.

sample of the workers at that time, and there was evidence that the men's nutritional state tended to deteriorate during their employment.[12] Since then the rations had been considerably improved, mainly by adding fish and providing more vegetables. The rations at the time of the present study were, however, still deficient in calcium, vitamin A, and (if cooking losses were considered) ascorbic acid, and contained an unduly high proportion of maize (Table II).

Even so, the nutritional state of the men examined was considerably better than that of the comparable group examined in February 1958, except in respect of dyssebacea and folliculosis which are signs of doubtful significance (Table III). It is considered unlikely that differences of the degree and range noted can be ascribed solely to seasonal factors, and it is believed that they probably reflect the improvements made in the rations.

TABLE III. PREVALENCE OF SIGNS OF MALNUTRITION AT COMPANY B IN 1958 AND IN 1960

Sign of malnutrition	*Prevalence %*	
	*February 1958 (36 men)**	*May 1960 (40 men)*
1. Leanness (arm skinfold thickness under 5 mm.)	61·1	42·5
2. Underweight (relative weight under 90%)	72·2	57·5
3. Lip abnormalities† (other than minimal abnormalities)	88·9	27·5**
4. Gum abnormalities† (other than minimal abnormalities)	72·2	35·0**
5. Tongue abnormalities† (other than minimal or mild abnormalities)	52·8	2·5**
6. Skin xerosis, trunk (marked xerosis only)	75·0	17·5**
7. Phrynoderma, trunk	2·8	2·5
8. Follicular enlargement, trunk	30·6	10·0
9. Dyssebacea/folliculosis (moderate or marked abnormalities only)	8·3	60·0**

These figures relate to men aged 20 - 39 and employed for at least 4 months.

* Figures derived from a study by Abramson, Slome and Ward.[12]

** Statistically significant difference.

† Ratings based on the nature, degree and extent of the signs found.[12]

Companies C - F

These 4 companies are grouped together, since there were only minor or inconsistent differences between the nutritional findings in each case. At each company, the men showed more signs of malnutrition than at company A.

The men at company C were housed in a compound, and for 5d. were provided with a midday meal, which cost about 1s. 6d. to prepare. The majority of the men were, however, dissatisfied with this meal, and did not think it worth the price. Only 35% took it regularly. At company D, the men were housed in a compound but not provided with food. Before a dispute with the management in the middle of 1959, they had been fully fed by the company. Currently, they received an extra 3s. a week in lieu of food. Their food purchases amounted, on average, to 20s. 6d. a week, according to their statements. This was possibly an overestimate. The men at company E were not housed at work. Most (68%) lived in hostels, 16% with their families, and most of the others in rented rooms. They were provided with a free vegetable soup at midday. Many were, however, dissatisfied with this soup, and did not take it at all. The men at each of these 3 firms (C, D and E) consumed meat, on average (median figures) on 2 days a week, milk (as a beverage or with porridge) on 0 or 0·5, green or yellow vegetables on 0 or 1, and fruit, fish, eggs, and *amasi* on 0.

The men at company F had a slightly better diet. These men too were not housed at work. Most (52%) lived in rented rooms, 18% with their families, and 30% in hostels. For 6d. they were supplied with a midday meal, costing about 1s. 6d. to prepare, which 75% of them took regularly. This was the best-paid group. They took meat, on average (median figures) on 4·5 days a week, milk (either as a beverage or with porridge) on 2, and green or yellow vegetables on 2·5, but fish, eggs, fruit and *amasi* on 0. In spite of the slightly better diet, there was no convincing difference between their nutritional state and that of

the men at companies C, D and E, taken severally or together. This may be partly explained by the fact that they were slightly older. Their mean age was 31·9 years, compared with 29·1, 26·1, and 27·3 years at companies C, D, and E respectively.

Meal Habits

The men who were not fully fed at work were asked what food they usually took (when on day shift) before starting work. There were 27% who took no food at all, 8% who had tea only, 59% who had a cereal food, with or without tea, and 6% who had a more substantial breakfast. During working hours, only 3 men (2%) had mineral drinks at least once a week, and only 11 men (7%) took cakes or buns at least once a week.

The men at company D were questioned more fully about their usual eating habits during the day. Although these men are not provided with food by the company, certain foods are available for purchase at or near the factory. It was found that 45% took no food at all before starting work, 2% had tea only, 48% had a cereal meal, and only 5% had a more substantial breakfast. During working hours, 48% took only a cereal food, with or without tea, while 52% had meat, fish, vegetables or *amasi* as well. From the time they awoke until they left work, there were 45% who consumed nothing but cereal foods, with or without tea; some of these (18% of the total) had no breakfast.

Attitudes to Food provided by Management

There was considerable variation in the men's attitudes to the food provided by the respective companies. At company A they were satisfied with the changes in their rations, and only 18% had complaints about the food, its preparation, or serving. At company B also, the men approved of the changes in their rations, but 70% had complaints about their food. At company D, where company feeding had been stopped, the men did not favour its resumption, apparently because of the monotony of the meals previously served. At company E, 95% were dissatisfied with the soup offered them, less because it was unsatisfactory than because they would have preferred a thicker dish, such as a stew. At company C, 82% were dissatisfied with the midday meal provided, and few took it regularly. At company F, where the midday meal was similarly priced, 68% had complaints, but most took the meal regularly.

The usual grounds for complaint were the unpalatability of the food offered, the poor quality of the ingredients used, and the small quantities of meat and beans supplied.

Nutritional Knowledge

A series of questions revealed that the men's standard of knowledge about the relationship of food to health was unsatisfactory. For example, 57% thought that *phuthu* (thick maize porridge) was healthier than bread. Only 51% considered potato to be healthier than mealie rice (a refined maize product), and only 58% regarded *amasi* (sour milk) as being healthier than *mahewu* (a sour maize gruel).

The men at company A, who had been issued with daily vitamin pills for 3 weeks, were asked what the pills were for. As many as 55% said they did not know.

DISCUSSION

The men were obviously a malnourished group. Although many of the abnormalities noted were in themselves slight, and of kinds which might be related to causes other than general malnutrition, the concurrence in most of the men of a wide variety of abnormalities made non-nutritional explanations unlikely. Other studies have shown a similarly high prevalence of malnutrition among African workers in Durban.[18, 19]

The findings were consistent with the known effects of a predominantly maize diet similar to that of these workers, and were of a kind frequent among maize-eating populations.[20,21] The men's malnutrition was probably primarily dietary in origin, and related to their low consumption of animal tissues, milk, yellow and green vegetables, and fruit. Whatever the extra stresses to which the men were exposed, such as infestation and severe exertion, their diet appeared to be qualitatively inadequate to meet their needs.

Many of the signs of malnutrition are chronic and irreversible, so that our findings may in part be related to the diet and circumstances of the men before their current employment. Other of the signs found are, however, readily reversible by dietary modifications. It can thus be concluded that the condition of the men can to a considerable extent be ascribed to their current diet and circumstances. This conclusion is supported by the wide variations found among the 6 groups studied, by the occurrence of changes in the condition of representative workers at company B following the modification of their diet, and by the evidence previously found, at the same company, of a deterioration during their employment in the condition of men on a poor diet.[12]

The high prevalence of signs of malnutrition indicates that most of the men were in a sub-optimal state of health, which might affect their working efficiency. In a controlled study at a Californian aircraft factory, an association was found between the administration of vitamin supplements and a rise in efficiency together with a drop in rates of absenteeism and labour turnover.[22] Although there are few controlled studies indicating a direct relationship between nutritional state and work output on the job, there is considerable evidence suggesting that better feeding may enhance productivity.[23] On various Witwatersrand mines, for example, it has been found that the introduction of mid-shift feeding has been followed by improved performance, a reduction in accidents, or a drop in illness and accident absenteeism.[24] Similarly, a Transvaal foundry reported a 40% rise in production and a drop in labour turnover, within 4 months of the introduction of mid-morning and midday meals.[25] The managements of companies C and D commented that their workers appeared to tire easily during the day. At company B the management reported that the improvements in the men's rations since 1958 had been accompanied by a rise in productivity and a drop in absenteeism, the latter falling from 8·2%[12] to 4%; this could not, however, be regarded as a controlled observation, since there had been other concurrent changes in management policy.

PRACTICAL IMPLICATIONS

Clearly, there are many possible approaches to the problem of malnutrition among African workers in South

Africa.[26, 27] A most important contribution, however, and one which we wish to emphasize, is that which can be made by employers and employers' organizations. Apart from the provision and more effective utilization of industrial health services, 3 allied measures can be recommended — provision of good food, increased wages, and health education. It is encouraging that these recommendations have been adopted, in whole or in part, by all 6 of the companies studied.

1. *Provision of Good Food for Employees*

Where workers are housed and fully fed by the company, the management should ensure that their diet is nutritious. Competent advice should be obtained on its planning, and steps taken to ensure that the food is attractive to the workers. In this regard, the experience of company B provides an object lesson. Not only was the poor diet of 1958 associated with a high prevalence of malnutrition, but discontent with the food was at that time considered to be a possible contributory factor to the men's low morale and productivity.[12] It is not difficult to plan a diet which is both nutritious and palatable. At companies A and B dietary changes aimed at increasing the nutritional value of the rations have been welcomed by the workers. The cost of such a diet need not be excessive; the food provided by these companies cost the managements, we are informed, under 7s. and 7s. 9d. per person a week, respectively.

Our finding that the men at company A, who were housed and fully fed at work, had less evidence of malnutrition than the other groups, does not imply that housing men in barracks and providing them with good food can, in general, be regarded as a satisfactory solution. The separation of men from their families carries hazards. As has been said, 'the social, medical, economic and moral results of this migratory labour are catastrophic, and the system cannot be condemned too strongly'.[28]

Where men are not fully fed at work, they should be provided with meals during working hours, either free or at a low price, the main burden of cost falling on the company. Such meals, if reasonably palatable, are likely to be well received. At company F, although many of the men were not completely satisfied with the meal, most of them took it daily, at the price of 6d. At company C, where there was more dissatisfaction with the food, few men took it regularly, although it was priced at only 5d. The workers should be consulted and it is essential to take their preferences into account. At company E, where the men would have liked a stew, they were offered free soup, which few of them took. It is noteworthy that at 3 of the companies studied, the managements had very little idea of the men's views of the food provided for them. Fortunately, the comments of the men questioned indicate that the preferences of African labourers are not inconsistent with the planning of sound meals. For example, they frequently expressed a wish for meat and vegetable stews and for beans, which can make a valuable contribution to their diet. *Amasi*, similarly, is both nutritious and acceptable, and can be cheaply prepared from skim-milk powder. The provision of acceptable food may help to promote sound management-employee relations, and contribute to good morale.

Consideration should be given to the provision of a breakfast or mid-morning snack, particularly as many workers take little or no food before work. At certain companies, for example those handling food, the free issue of fruit or other good foods should present few problems.

Where there is a company canteen selling food to the workers, it is important to ensure that nutritious foods are available. It is noteworthy that, contrary to the usual belief, relatively few workers appear to partake of mineral drinks, cakes, and buns during working hours. It is likely that among better-paid workers there is a higher consumption of these items.

The advisability of a vitamin supplement is questionable. Where adequate food can be provided, this is probably preferable. Few men will indefinitely continue taking a tablet daily, particularly when, as at company A, they are ill-informed as to its nature and purpose.

2. *Increased Wages*

Apart from the provision of good food at work, there is a strong case for the raising of wages. The worker's home diet is of considerable importance. The findings at company F, where men were provided with a nutritious and acceptable midday meal, but were not in a better nutritional state than other workers, possibly illustrate the importance of this factor.

The mean weekly cash wages of the men not fully fed at work was £2 19s. 11d. Of these men, 66% were married, with an average of 2·5 living children. The wives of only 2% were gainfully employed. The cost of the 'minimum diet' of an African family of man, wife and 3 children, as recommended by the Department of Nutrition, Pretoria (cited by de Gruchy[29]) is £3 0s. 1d. per week, excluding the recommended addition of fruit (calculations based on average Durban retail prices in December 1959[30]). These figures speak for themselves. Under such conditions, malnutrition must remain a major contributory factor to morbidity and mortality in this country.

Most of the companies studied had either recently effected, or were contemplating, wage increases. Doubt was, however, expressed whether these increases would benefit the health of the men or their families. 'The workers won't spend more on food,' was one management's prediction. Our findings have demonstrated that the knowledge of many workers about the relative value to health of various foods is defective. Concomitant health education is therefore of considerable importance, since without it wage increases may have only a limited effect on health.

3. *Health Education*

It is not easy to alter people's food habits. However, trained health educators, who can produce valuable improvements in living habits and health,[31,35] have an important potential rôle in industry in this country. Such personnel should have little difficulty in improving workers' dietary knowledge, in modifying their habits of food buying, preparation and consumption, and in motivating them to form bulk buying clubs. At one Durban factory, health educators working in conjunction with the factory medical officer were able to produce a 100% conversion from white to brown bread consumption.[36]

Where a company contemplates changes in the provision of food or meals to its workers, such changes could use-

fully be preceded and accompanied by a planned programme of health education, to ensure their positive acceptance by the workers. Failing such a plan, changes, though well-intentioned, may produce discontent.

Few trained health educators are available as yet, and it is not realistic to suggest that one should be employed by every company. However, in view of the many ways in which such personnel could improve the health of workers and their families,[37-40] we recommend that health educators should be employed by industrial and other employers' organizations, for work in groups of companies.

SUMMARY

A study was undertaken in May 1960 of the nutritional state and diet of low-paid African labourers, aged 20 - 39, predominantly migrant, and employed at 6 companies in or near Durban.

Many varied signs of malnutrition were found. Evidence is presented that this could to a considerable extent be ascribed to the men's current diet and circumstances.

The workers' standard of nutritional knowledge was poor.

The practical implications of the findings are discussed, with special reference to the rôle of employers and employers' organizations. Three allied measures are recommended: the provision of good food for employees, wage increases, and health education.

We are indebted to the managements and men for their interest and cooperation and to the following personnel for their assistance: Sister T. Triegaardt; health educators A. T. Mbombo, J. Mngadi, M. Ngidi, W. S. Nkuhlu, R. Philips and I. I. Pitso; medical recorders W. H. Pietersen and S. J. Maharaj; and nurse-aide E. Handley. Dr. S. M. Joubert, Department of Pathology, University of Natal, performed the serum-protein estimations.

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COENZYMES I AND II IN THE HUMAN EPIDERMIS*

CHANGES IN NUTRITIONAL SUN-SENSITIVITY (PELLAGRA) AND DURING CYTOSTATIC DRUG THERAPY

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The commonest sun eruption in South Africa is pellagra. Although a niacin deficiency in pellagra is clearly not the whole story, it suggests a means by which nutritional and photodynamic influences may reinforce one another.

Nicotinamide is known to be built by cell nuclei into the pyridine nucleotides, coenzymes I (DPN) and II (TPN), which are essential in the cellular respiratory chain. These coenzymes are not only altered by ultraviolet light, but are influenced by free radicals resulting from the action of light. Other parts of the respiratory chain are also influenced by diet, light and sun-sensitizing drugs. It therefore seems that altered intracellular respiration may offer a pathway for the action of light in vitamin-deficiency states, thus bringing about abnormal changes in epidermal cells.

Since nucleic acid synthesis is affected by cytostatic drugs, a few observations were made to test their action on pyridine nucleotide synthesis in the epidermis. These are also briefly reported, although the changes are obscure.

METHODS

Only the oxidized phosphopyridine nucleotides were assayed. These represent DPN and TPN, coenzyme III, and such precursor substances as also yield fluorescent products in the technique used. No enzymatic separation of these components was attempted.

Skin assays were made from samples of whole-thickness epidermis removed with a 3 mm. Volkmann spoon under procaine anaesthesia. These epidermal samples were blotted gently in the spoon, removed as a 'plug', weighed wet without mincing, and homogenized at once. Roughly 10 mg. epidermis could be obtained from 1 square cm. of

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skin. Oxalated venous blood was used for the assay on whole blood. All assays were performed promptly on collecting the specimens.

The method used was the methyl ethyl ketone fluorimetric assay of Kaplan and Ciotti,[1] adapted to the Eel fluorimeter. The fluorimeter was calibrated over the range 0 - 4 μg. DPN, using DPN (Sigma Chemical Co.) of strength assayed independently by the cyanide addition procedure[1] on the ultraviolet spectrophotometer. Quinine sulphate in 0·1 N. H_2SO_4 was then used as a stable substitute for the DPN standard in concentrations yielding equal fluorescence. Duplicate assays were made throughout and were usually within 0·6 μg. per g. or 0·6 μg. per ml. or less, of one another.

RESULTS

Assays of oxidized pyridine nucleotides in skin and/or blood were performed on 48 patients, the results of which appear in the tables. All blood values are expressed as equivalents of DPN in μg. per ml., corrected to a hematocrit of 45%. Skin values are likewise equivalents of DPN in μg. per g. moist weight.

Blood Values

In White and Bantu patients without pellagra (cases 1 - 23), the blood values of pyridine nucleotides in the Bantu patients were on a rough average 25% lower than those in White patients, though still within the accepted normal range. The lowest values were found in 3 Bantu patients (cases 21 - 23) who were sick but showed no pellagra. The arithmetical average of the Bantu values (cases 7 - 23) was about 30 μg. per ml., while the average in a few White patients (cases 1 - 6) was 40 μg. per ml. In 13 Bantu patients with pellagra (cases 24 - 28, 33, 35 - 41) who were not critically ill and who are presumed to have had no vitamin treatment, the average value was 34 μg. per ml. Making allowances for the sampling errors in both groups, one may say that the blood values in the Bantu subjects are not only roughly normal, but are also roughly the same whether they suffer from pellagra or not.

Skin Values

The skin values of pyridine nucleotide showed a greater range of variation. In 7 White patients who had had no vitamins (cases 1 - 6, 47) the values lay between 36 and 120 μg. per g. In one non-pellagrous Bantu subject studied (case 48) the values also fell largely within this range. No other non-pellagrous Bantu cases were studied, if we exclude the values obtained on the unaffected skin

TABLE I. NORMAL AND ABNORMAL SKIN AND BLOOD VALUES OF PYRIDINE NUCLEOTIDES FROM WHITE PATIENTS

Case	*Test*	*Sex*	*Age*	*Blood (μg./ml.)*	*Skin (μg./g.)*	*Remarks*
1	a	F	47	40·9	36·1	Atopic eczematous skin, back.
	b				36·0	Normal skin, back.
2		F	18	54·9	110	Normal skin, shoulder.
3		M	29	41·7	67	Normal skin, back.
4		F	52	37·6	70·5	Normal skin, arm.
5		F	51	37·4	64·0	Normal skin, shoulder.
6	a	M	77	34·6	61·0	Non-scaly psoriatic skin.
	b				14·5	Dry, exfoliated, psoriatic scalp scales.

TABLE II. BLOOD LEVELS OF PYRIDINE NUCLEOTIDES IN NON-PELLAGROUS BANTU SUBJECTS

Case	*Sex*	*Age*	*Blood (μg./ml.)*	*Diagnosis and remarks*
7	F	25	36·0	Pityriasis rubra pilaris.
8	M	8	36·2	Pityriasis rubra pilaris.
9	M	31	30·9	Stomatitis — ? type.
10	M	16	28·1	Lichen planus.
11	M	10	25·0	Eczema.
12	F	50	33·5	Lues III.
13	F	31	35·4	Thyrotoxicosis.
14	F	40	33·8	'Lodger'.
15	M	45	36·3	Seborrhoeic eczema.
16	F	14	28·2	Otitis externa.
17	F	40	30·9	Cardiac failure.
18	F	28	43·2	Not pellagra.
19	M	16	29·9	Not pellagra.
20	M	24	25·4	Colles fracture.
21	F	31	15·2	Gastro-enteritis — ? type.
22	M	45	17·4	Pneumonia — convalescent.
23	F	21	21·8	Acute salpingitis.

TABLE III. BLOOD LEVELS OF PYRIDINE NUCLEOTIDES IN PELLAGROUS BANTU SUBJECTS

Case	*Sex*	*Age*	*Blood (μg./ml.)*	*Remarks*
24	M	40	28·3	Untreated pellagra with dementia.
25	M	26	32·1	Untreated pellagra with dementia.
26	F	45	31·5	Pellagra with Riehl-like picture. No vitamins given for past 3 months.
27	M	19	26·4	Untreated pellagra. Peri-oral 'mudpack'.
28	F	30	28·8	Untreated pellagra.
29	M	8	38·2	Active pellagra, possibly treated.
30	F	27	49·3	Active pellagra with dementia, under treatment.
31	F	30	55·5	Active pellagra, under treatment.
32	M	40	53·7	Subsided pellagra, under treatment.

TABLE IV. PYRIDINE NUCLEOTIDE LEVELS IN THE BLOOD, AND THE NORMAL AND ABNORMAL SKIN FROM PELLAGROUS BANTU SUBJECTS

Case	*Test*	*Sex*	*Age*	*Blood (μg./ml.)*	*Pellagrous skin (μg./g.)*	*Adjoining non-pellagrous skin (μg./g.)*	*Remarks*
33		M	43	33·8	63	68	Non - keratotic pellagrous skin from extensor surface, forearm.
34		F	42		42	49	Forearm skin.
35		F	20	41·9	32·5	53·4	Recent case. Forearm skin.
36		M	16	32·0	27·5	28	Forearm skin. Not keratotic.
37		F	10	35·9	27·9	31·1	Forearm. 1 month duration. Non-keratotic skin.
38		M	22	28·3	24	30	Pellagra with dementia. Forearm skin.
39		M	60	33·4	36	49	Ordinary pellagra.
40		F	39	50·7	34·8	40·9	Pellagra. 3 months' duration.
41	a	M	42	48·5		29·8	Extensor surface forearm.
	b					30	Flexor surface forearm.
42		M	45	47·6	36·1	37·9	Pellagra dementia.
43		M	35		191	219	Several weeks' vitamin therapy. Skin lesions very evident.
44	a	M	?35	39·4	168	169·4	Several hours' intravenous vitamin-B complex administration. Pellagra with encephalopathy.
	b			45·3	133·2	121·3	5 days later. No further intravenous vitamin therapy. Skin lesions still evident.

TABLE V. BLOOD AND SKIN PYRIDINE NUCLEOTIDE VALUES FROM PATIENTS UNDERGOING TREATMENT WITH CYTOSTATIC DRUGS

Case	Test	Sex	Age	Race	Blood (μg./ml.)	Skin (μg./g.)	Remarks
45	a	M	62	W	33·7	100	Cancer of stomach. Gastrectomy 10 days before with blood transfusion and vitamin-B complex. Shoulder skin. No cytostatics.
	b				33·8	89·6	After 1 week 5-fluorouracil. Treatment produced mouth ulcers. Shoulder skin.
46	a	M	50	W	31·3	178·3	Cancer of pancreas. Vitamins up to 1 week before. Shoulder skin.
	b				35·7	183·5	After 5 days' treatment with 5-fluorouracil. No side reactions. Shoulder skin.
47	a	F	72	W	29·2	120	Hodgkin's disease. Before endoxan treatment. No vitamins. Shoulder skin.
	b				35·6	79·3	After 5 days' endoxan treatment.
	c				36·5	33·5	After 9 days' endoxan treatment.
	d				40·1	58·4	After 11 days' endoxan treatment.
	e				38·3	62·1	After 16 days' endoxan treatment.
	f				31·1	35·7	After 20 days' endoxan treatment.
48	a	M	19	B	39·2	58·3	Hodgkin's disease. Before endoxan treatment. Shoulder skin. No vitamins. Ward diet, as given at non-European hospital.
	b				45·0	84·2	After 5 days' endoxan treatment.
	c				40·6	90·4	After 10 days' endoxan treatment.
	d				34·5	144·6	After 16 days' endoxan treatment.

of pellagrous Bantu subjects. The untreated pellagra patients (cases 33 - 42) showed generally low values in the skin by comparison, although values which did not differ greatly were found in non-pellagrous White subjects (cases 1a and b, 47c and f) as well. Comparing the levels in pellagrous skin with those of the unaffected skin nearby in the same subject, the pellagrous skin showed values about 15% less than the normal skin. This was probably not fortuitous, though one could ascribe it to dilution of the sample by scale or oedema, or variations according to site. The following observations suggest that the difference is important. Thus, case 1 (a and b) showed the same value for eczematous and non-eczematous skin; case 6 showed a normal value in abnormal skin; psoriatic scale (case 6b) despite dessication showed values of a quarter of the wet weight of whole epidermis with little scale (case 6a); case 41 (a and b) showed that the aspect of the forearm chosen for sampling did not influence the value obtained. Moreover, many of the pellagrous lesions were not notably scaly. From these considerations we may say that the skin value of pyridine nucleotides is low in pellagra, and lower in pellagrous skin as opposed to uninvolved skin in the same person, but that a low value as such is found in non-pellagrous subjects as well where there is no reason to suppose that they will develop pellagrous dermatitis.

The present study offers a few indications regarding the rate, extent and ability of the skin to synthesize pyridine nucleotides. Firstly it appears that severe illness does not interfere with the ability to use nicotinamide for nucleotide synthesis, and abnormal skin may do this without difficulty (cases 6 and 46). Secondly, case 46 shows that the levels readily rise above normal values on giving vitamin-B complex. Thirdly, cases 43 and 44 show that these high values are obtained in the skin in pellagra without any difficulty after nicotinamide administration, and that no obvious block in nicotinamide utilization exists. Fourthly, the same cases show that the skin can be loaded with more than sufficient vitamin within half a day if necessary, but that the lesions may still take a few weeks to heal.

Cytostatic Drugs

Four experiments were made to see if cytostatic drugs influenced the level of pyridine nucleotides in blood and skin. 5 - fluorouracil in cases 45 and 46 appeared to have no influence despite toxic action on the oral mucosa in one (45b). Endoxan is known to affect the epidermis greatly in certain cases,[2] though it failed to do so visibly in cases 47 and 48 which were investigated in this study. The blood levels showed a slow rise and fall over a 3-week period in both cases but the skin changes could not be superimposed in the 2 cases, and the fluctuations observed are still obscure.

SUMMARY

Oxidized pyridine nucleotides which contain nicotinamide were assayed in the skin and blood. The values in the solar dermatosis of pellagra indicate that the level drops slightly in pellagrous skin and is generally lower than normal in this type of malnutrition. However, the ability to synthesize these coenzymes was evidently still normal, though the onset and cure of a pellagrous dermatosis cannot be clearly linked to the level of niacin-containing coenzymes in the skin. Cytostatic drugs influence the coenzyme levels in ways that cannot be interpreted at present.

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SOME THOUGHTS ON THE LACRIMAL CANALICULI*

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Epiphora may result from inadequacy of the canalicular passage anywhere along its course. Although a commoner source of epiphora lies in the diseased lacrimal sac and naso-lacrimal duct, the problem of malfunction of the canaliculus occurs sufficiently often to merit considerable thought. This paper deals particularly with 3 sites of canaliculus inadequacy or interference, and with certain conditions at these sites.

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EVERSION OF THE INFERIOR PUNCTUM, WITH OR WITHOUT ITS OCCLUSION

Before investigating the more difficult causes of epiphora in a patient it is important to establish both the patency and proper position of the inferior punctum. Where the punctum is chronically everted, associated chronic conjunctivitis may cause its occlusion. In such a case, intensive topical antibiotic therapy, followed shortly by cautery puncture to the conjunctiva adjacent to the punctum, is usually a most effective combined procedure.

Cautery Puncture

The only local anaesthetic required for this is the application to the area of 4% cocaine drops mixed with adrenaline 1 : 1000. Occasionally it may be necessary to apply solid cocaine crystals to the conjunctiva especially if the eversion is fairly well-marked.

Approximately 6 - 8 applications of the red-hot electrocautery point are made to the conjunctiva around the punctum, mainly inferiorly and inferolaterally. These penetrate for approximately 1 mm. and care must be taken not to involve the canaliculus, which in this part of its course is superficial. Frequently this procedure dramatically cures an elderly patient with a chronic dribbling, dirty, eye and raw, exposed palpebral conjunctiva on the medial aspect of a sagging lower lid.

The punctum may have been exposed for so long that it is extremely stenosed. It may then require a modified 3-snip operation to make it function. However, replacing the punctum in its proper position, where it snuggles close to the globe and can hardly be seen, produces a remarkable effect on the hardened, previously exposed punctum. I feel that the vast majority of puncta with varying degrees of stenosis respond to this treatment and in only a few cases will a 3-snip operation be necessary. The extent of the 3-snip operation should be restricted.

The cautery punctures may have to be repeated on occasion and massage with lanolin or zinc-cream ointment into the thickened taut skin of the lower lid may be required as an adjunct in certain cases. In most cases of eversion of the punctum of the lower lid as above described this treatment will prove satisfactory.

Where there is grosser ectropion with eversion of the punctum a deliberate plastic procedure by an ophthalmologist is indicated.

INTERRUPTION AND OBSTRUCTION IN THE INFERIOR CANALICULUS

This may cause the surgeon many long hours of frustration. Here, prevention of scarring of tissues in malposition is most important. Trauma is the commonest cause of inferior canalicular obstruction.

Canaliculoplasty by Primary Suture of a Recent Injury

The following is a case-report of an accidental injury in which this procedure was used effectively:

> After a head-on collision on the cricket field, the patient (Mr. O.Q.S.) was unconscious and suffered a severe facial laceration. His right lower lid was almost entirely torn away. An extensive and deep laceration traversed the medial canthus, proceeded across the lid laterally through layers down to the fornix, and then continued from the outer canthus down on to the cheek. This left the lid lying near his ear and attached only by a small amount of tissue.
>
> The inferior canaliculus was reconstituted by the method to be described, together with extensive repair of the other lacerations. The patient recovered well with no epiphora. However, the deep-lying wound in the fornix contracted and an area of exuberant granulation occurred in the conjunctiva over the fornix. The bands were later freed and the granulations excised. Then a mucous membrane graft was made to the area and a skin and muscle operation was performed. Four months after the last procedure no epiphora was present.

Laceration of the inferior canaliculus below the medial canthus frequently occurs in a wide variety of injuries to the lower lid. These include assaults with the fist, stab-wounds, motor accidents, and a hanger hooking the lid (in a department store sale). I have treated many patients with such injuries in the manner to be described. Very often these patients are treated in the acute stages by casualty officers or junior residents, who do not appreciate the full extent of the injury. They usually know nothing of the resultant watering eye, nor of the cosmetic blemish of the ectropion of an inadequately-repaired lower lid.

The laceration invariably leaves the lateral portion of the torn inferior canaliculus held fast in the firm surrounding tissues of the lid with the cut end gaping. The medial portion shrivels up into the mass of ripped tissues and is amazingly difficult to identify. This may be impossible even with the injection of dye or milk *via* the upper punctum. If the medial portion of the canaliculus can be identified then a fine suture should immediately be attached to it. A 2 mm. polythene tube with a specially-made stylus (Fig. 1) should be passed *via* both portions of the canaliculus into the nose *via* the nasolacrimal duct.

Fig. 1. View of end of stylus in polythene tubing (enlarged).

I have found that the method first suggested by Greaves,[1] and since modified, most satisfactory. He described the retrograde passage of a cannula, containing blue nylon thread, along the canaliculus. Our theatre sister has modified Dr. H. J. van der Merwe's[2] idea and has made a fine probe which is inserted through polythene tubing. This probe may be used instead of the cannula and sometimes it is preferable.

With the patient under general anaesthesia, the probe or cannula is passed through the upper canaliculus. A curve is fashioned in the end of the instrument so that it may negotiate the junction of this canaliculus with the common canaliculus. Often the lower and upper canaliculi join in such a manner that no difficulty is met in passing the instrument, as described, into the medial portion of the torn lower canaliculus. Occasionally considerable difficulty is experienced, so much so that the sac may have to be opened and the canaliculus negotiated through it. Once the instrument is through the medial portion of the canaliculus no difficulty is found in bridging the gap with the tube going through the lateral portion of the canaliculus and out through the lower punctum. The procedure is an arduous one and may take hours to complete.

Once the gap has been closed, the probe or cannula is carefully withdrawn leaving the tube of nylon *in situ*. The tissues external to the canaliculus should be carefully sutured with 6 - 0 black silk on 81 - 7 Grieshaber needles. Then a deep vertical mattress suture (0 black silk) is inserted and should slightly overcorrect the medial canthus. The skin is sutured with 4 - 0 black silk. The strong mattress suture is important in preventing the wound from gaping — this marked tendency indicates the need for overcorrection.

The polythene tube or nylon should be well strapped to the forehead or cheek and a retaining suture should be placed near the inferior punctum and through the tube. This should be kept *in situ* for 6 weeks if possible. A careful watch must be kept to see that the punctum does not become torn through.

This procedure should be performed within 36 hours of the trauma — the sooner the better. Results have been encouraging. I have not kept figures, but success has frequently been achieved. It is interesting to note that the superior punctum is not at all efficient in maintaining the drainage of tears.

Once a stricture has occurred then a variety of plastic procedures, with or without mucous membrane grafts, will have to be attempted. These results do not compare with those of primary procedures.

OBSTRUCTION IN THE MEDIAL END OF THE CANALICULI

The last 2 - 3 mm. of the common canaliculus, before it enters the lacrimal sac, are affected here. The lesion often

involves the related sac wall. If the canaliculi enter the sac separately then they may each be affected by the condition. The occlusion may be complete or nearly so. Microscopic examination of such a lesion, which I removed in London some years ago, revealed a non-specific chronic inflammatory change.

Probing with the lacrimal cannula during irrigation and confirmation on X-ray examination with radio-opaque material will identify the condition. Partial occlusion of the canaliculus together with obstruction in the nasolacrimal duct may result in a ball-valve effect on the lacrimal sac, particularly at the time of irrigation; this would cause a distended sac.

Operative Procedure

The approach is similar to that made for dacryocystorhinostomy, except that the incision is placed slightly more laterally than the usual one on the nasal side of the medial canthus. The canaliculi at this deep level are found just under the deep fibres of the medial ligament, so care must be taken when dividing the ligament. A probe, which is inserted along the inferior and common canaliculi, is isolated, and the lesion is then identified. The obstruction is excised and the remaining canaliculus is invaginated into the elliptical gap which has been fashioned in the lateral wall of the sac. Sutures of 6-0 black silk on 81-7 Grieshaber needles maintain the position of the canaliculocystic junction. This is invariably a very protracted procedure and it is essential that complete haemostasis is obtained in the surgical field.

This haemostasis may be obtained by positioning the patient in the anti-Trendelenberg position and using diathermy coagulation to any bleeding points. Fluothane anaesthesia assists in attaining the ideal. Because of the usually protracted nature of the surgery I have found it better to avoid the use of hypotensive anaesthesia, particularly in elderly patients, except as above described. Occasionally I have used more intensive hypotensive anaesthesia, especially when surgery has already been performed in the area.

While the sac is open, the patency of the nasolacrimal duct must be established by irrigation. If the passage is not patent then a dacryocystorhinostomy must be performed there and then.

Although not many patients have had the above described 'canaliculo-dacryocystorraphy' to date, the results are encouraging, especially if the polythene tubing, which is left *in situ* in the canaliculus, can be maintained for several weeks.

Epiphora, especially in younger active people, is sometimes very incapacitating, and I feel that the long hours, often late at night, which may have to be spent completing some of the abovementioned manouevres, are well spent if the watering eye is cured or, better still, prevented.

SUMMARY

Three sites of possible inadequacy of, or interference with, the lacrimal canaliculi are discussed.

Operative procedures to correct conditions arising at these sites are described.

The photograph was taken by Mr. D. Smith, of the Department of Surgery, University of the Witwatersrand, to whom I tender my thanks.

REFERENCES

1. Greaves, D. P. (1958): *Eye Surgery*. p. 295. Bristol: John Wright (quoted by Stallard, H. B.).
2. Carter, M. and van der Merwe, H. J.: Personal communication.

43ste MEDIESE KONGRES (M.V.S.A.), KAAPSTAD, 24-30 SEPTEMBER 1961 : 43rd MEDICAL CONGRESS (M.A.S.A.), CAPE TOWN, 24-30 SEPTEMBER 1961

SCIENTIFIC EXHIBITIONS

A circular letter from the Chairman of the Scientific Exhibition Sub-Committee is reproduced below for the information of members of the Association:

'It it the intention of the Organizing Committee to arrange a scientific exhibition in conjunction with the proceedings of Congress. We feel that this will offer a unique opportunity for the demonstration of scientific research and other work presently being carried out in the Union, not only by official bodies and sponsored research units and groups, but also by individual doctors and scientists.

'The approach to this exhibition will be as broad as possible and will include anthropology and genetics, psychology and industrial aptitude testing, public health and sanitation, the medical applications of atomic energy, nutrition, food technology and quality control, therapeutic substances, production and control, health education and immunization procedures, veterinary work and the zoonoses, hospital planning and medical services administration, the history of medicine and its instruments, tribal medicine and customs, as well as the full field of medical research. General practitioners are included.

'One part of the exhibition will be a cinematograph theatre showing a daily programme of medical and scientific films, coloured slides, etc. It is hoped that closed-circuit television will be included.

'This preliminary circular is being sent to as many organizations and individuals as possible who may care to take part in the exhibition. To assist the planning committee, it would be appreciated if prospective exhibitors would indicate, as soon as possible, whether or not they or their organizations would be prepared to exhibit, and if so briefly indicate:

'1. The nature and title of the exhibit.
'2. Whether it will be (*a*) static display, (*b*) working demonstration, or (*c*) ciné or slide projection.
'3. Approximately what (*a*) linear, or (*b*) square footage of display space will be required.
'4. Whether power, water, drainage, or gas will be required.
'5. If films or slides are to be shown, the approximate time taken.

'After this information has been received, preliminary plans will be drawn up and a subsequent circular early in 1961 will deal with the detailed plans, arrangements for demonstrators, delivery and erection, insurance, etc.

H. O. Hofmeyr
Chairman, Scientific Exhibition Sub-Committee

Medical House
35 Wale Street
Cape Town'

FARMASEUTIESE NUUS : PHARMACEUTICAL NEWS

SQUIBB LABORATORIES (PTY.) LTD.

Squibb Laboratories (Pty.) Ltd. took occupancy of new premises on 5 December 1960 at Electron Avenue, Isando, Transvaal, telephone 975-4614, P.O. Box 48, telegrams 'Ersquibb'.

As from 5 December the distributor-arrangement with Protea Pharmaceuticals Ltd. ceased, and all first-stage distribution of Squibb products will be effected by Squibb Laboratories from the above address.

All enquiries concerning Squibb products should be directed to the above address.

AMPTELIKE AANKONDIGINGS : OFFICIAL ANNOUNCEMENTS

GOEDGEKEURDE MEDIESE HULPVERENIGINGS

Hieronder verskyn die lys van goedgekeurde mediese hulpverenigings soos op 1 November 1960. Lede behoort hierdie lys vir naslaandoeleindes byderhand te hou.

L. M. Marchand
Medesekretaris

Plaza-gebou 28
Pretoria
18 November 1960

1. A.A. Mutual Medical Aid Society, P.O. Box 9595, Johannesburg.
2. Abercom Group Sick Benefit Society, P.O. Box 494, Port Elizabeth.
3. African Cables Medical Benefit Fund, P.O. Box 172, Vereeniging.
4. African Explosives Medical Aid Society, P.O. Box 1122, Johannesburg.
5. African Homes Trust Sick Fund, P.O. Box 93, Cape Town.
6. African Oxygen Limited Medical Aid Society, P.O. Box 5404, Johannesburg.
7. Afrikaanse Pers Beperk se Siekefonds, Posbus 845, Johannesburg.
8. Alex, Aitken & Carter Medical Benefit Society, P.O. Box 2636, Johannesburg.
9. Algoa Medical Aid Society, P.O. Box 369, Port Elizabeth.
10. Argus Medical Benefit Society (Cape Argus Branch), P.O. Box 56, Cape Town.
11. Argus Medical Benefit Society (Daily News Branch), P.O. Box 1491, Durban.
12. Argus Medical Benefit Society (Star Branch), P.O. Box 1014, Johannesburg.
13. Associated Employers' Medical Aid Society, P.O. Box 7462, Johannesburg.
14. A.T.I. Medical Aid Society, P.O. Box 5057, Boksburg North.
15. Babcock and Wilcox Medical Aid Fund, P.O. Box 545, Vereeniging.
16. Bakers Ltd. European Employees' Sick Benefit Fund, P.O. Box 692, Durban.
17. Bloemfontein Municipal Employees' Medical Aid Society, P.O. Box 288, Bloemfontein.
18. Boart and Hard Metal Products Medical Aid Society, P.O. Box 9325, Johannesburg.
19. Boksburg Municipal Employees' Medical Aid Fund, P.O. Box 215, Boksburg.
20. British General Electric Co. (Pty.) Ltd., Medical Aid Society, P.O. Box 9329, Johannesburg.
21. Broderick Medical Aid Society, P.O. Box 186, Vereeniging.
22. Building Societies Joint Medical Aid Fund, P.O. Box 5728, Johannesburg.
23. S. Butcher & Sons Ltd. Medical Aid Society, P.O. Box 1004, Durban.
24. Cape Portland Medical Aid Society, P.O. Box 1067, Cape Town.
25. Cape Times Medical Aid Society, P.O. Box 11, Cape Town.
26. Cape Town Municipal Employees' Association Medical Aid Society, P.O. Box 1939, Cape Town.
27. Central News Agency Ltd. Medical Benefit Society, P.O. Box 1033, Johannesburg (excluding Cape Town and suburbs, Durban municipal area, Johannesburg and Witwatersrand, and Port Elizabeth and Pretoria municipal areas).
28. Chamber of Mines Medical Aid Society, P.O. Box 809, Johannesburg.
29. Civil Service Medical Benefit Association, P.O. Box 176, Pretoria.
30. Commercial and Industrial Medical Aid Society, P.O. Box 958, Johannesburg.
31. Consolidated Glassworks Limited Medical Aid and Sick Benefit Society, P.O. Box 562, Germiston.

APPROVED MEDICAL AID SOCIETIES

The following is the list of approved medical aid societies as at 1 November 1960. Members should keep this list for reference.

L. M. Marchand
Associate Secretary

28 Plaza Building
Pretoria
18 November 1960

32. Corner House Medical Aid Fund, P.O. Box 1056, Johannesburg.
33. Coronation Medical Aid Society, P.O. Box 1517, Durban.
34. Crooks Bros. Ltd. Medical Benefit Fund, 301 Smith Street, Durban.
35. D.F.A. Medical Benefit Society, P.O. Box 610, Kimberley.
36. Dorman Long (P.E.) Medical Aid Society, P.O. Box 9010, Port Elizabeth.
37. Eastern Province Cement Co. Ltd. Medical Aid Society, P.O. Box 2016, Port Elizabeth.
38. E.P. Newspapers Medical Aid Society, P.O. Box 1117, Port Elizabeth.
39. Egnep Medical Aid Society, P.O. Penge, Transvaal.
40. Escom Cape Western Undertaking Medical Aid Society, P.O. Box 117, Cape Town.
41. Escom (N.C.U.) Medical Benefit Society, P.O. Box 30, Colenso, Natal.
42. Escom (N.S.U.) Medical Aid Society, P.O. Box 2408, Durban.
43. Everite Medical Aid Society, P.O. Kliprivier, Transvaal.
44. Federated Employers' Medical Aid Society, P.O. Box 666, Johannesburg.
45. Federation of Master Printers of S.A. Medical Aid Society, P.O. Box 4465, Johannesburg.
46. First Electric Medical Aid Fund, P.O. Box 3961, Johannesburg.
47. Friend Medical Aid Fund, P.O. Box 245, Bloemfontein.
48. General Mining (Associated Companies) Medical Aid Society, P.O. Box 1007, Johannesburg.
49. General Motors Medical Aid Scheme, P.O. Box 1137, Port Elizabeth.
50. Germiston Industries Medical Aid Society, 113 Pylon House, Human Street, Germiston.
51. Gledhow-Chaka's Kraal Sugar Co. Ltd. Medical Benefits Fund, P.O. Box 55, Stanger, Natal.
52. Goldby, Panchaud and Webber Medical Benefit Fund, P.O. Box 1172, Johannesburg.
53. Goldfields Medical Aid Fund, P.O. Box 1167, Johannesburg.
54. Greatermans Medical Aid Society, P.O. Box 5460, Johannesburg.
55. Hubert Davies Johannesburg Staff Medical Aid Society, P.O. Box 1386, Johannesburg.
56. Sir J. L. Hulett & Sons Ltd. Medical Benefit Fund, P.O. Box 248, Durban.
57. Hunt, Leuchars & Hepburn Ltd. (Transvaal Staff) Medical Aid Society, P.O. Box 47, Johannesburg.
58. I.C.T. Medical Aid Society, P.O. Box 7018, Johannesburg.
59. Iscor Medical Benefit Fund, P.O. Box 450, Pretoria.
60. I.W.S. Medical Aid Society, P.O. Box 6946, Johannesburg.
61. J.W. Jagger & Co. Ltd. Medical Aid Society, P.O. Box 726, Cape Town.
62. Johannesburg Board of Executors' Medical Aid Society, P.O. Box 271, Johannesburg.
63. Klerksdorp Munisipale Werknemers Siektefonds, Posbus 99, Klerksdorp.
64. K. & L. Timbers Ltd. Staff Medical Aid Fund, P.O. Box 9, Elandsfontein, Transvaal.
65. Koegas Medical Aid Society, P.O. Koegasbridge, C.P.
66. Krantzberg Mines Medical Aid Society, P.O. Box 18, Omaruru, S.W.A.

67. Kroonstad Munisipale Mediese Hulpvereniging, Posbus 302, Kroonstad.
68. Legal and General Medical Aid Society, P.O. Box 4870, Johannesburg.
69. Mail, Times & Express Medical Aid Society, P.O. Box 1138, Johannesburg.
70. Marley Floor Tile Medical Aid Society, P.O. Box 67, Nigel.
71. Masonite Medical Aid Society, P.O. Box 57, Estcourt, Natal.
72. Max Engineering Medical Aid Scheme, P.O. Box 174, Vereeniging.
73. Metal Box Company of S.A. Ltd. Medical Aid Society, P.O. Box 7752, Johannesburg.
74. Municipal Employees' Medical Aid Society (Durban), P.O. Box 625, Durban.
75. Natal Building Society Medical Aid Fund, P.O. Box 947, Durban.
76. Natal Coal Owners' (Durban Staff) Medical Aid Society, P.O. Box 281, Durban.
77. Natal Estates Sick Fund Benefit Society, P.O. Mount Edgecombe, Natal.
78. Natal Industries Medical Aid Society, P.O. Box 1300, Durban.
79. N.T.E. Staff Medical Aid Fund, P.O. Box 39, Pietermaritzburg.
80. National Industrial Credit Corporation Medical Aid Society, P.O. Box 8296, Johannesburg.
81. National Portland Medical Aid Society, P.O. Box 21, Claremont, C.P.
82. National Trading Medical Aid Society, P.O. Box 2762, Johannesburg.
83. Northern Assurance Co. Ltd. Medical Aid Society, P.O. Box 8615, Johannesburg.
84. Northern Medical Aid Society, P.O. Box 3437, Johannesburg.
85. Northern Rhodesia European Civil Servants, Medical Aid Society, P.O. Box R.W. 13, Ridgeway, N.R.
86. Norwich Union Life Insurance Staff Medical Aid Society, P.O. Box 1226, Cape Town.
87. Ore & Metal Medical Aid Society, P.O. Box 3548, Johannesburg.
88. Pietermaritzburg Chamber of Industries Medical Aid Society, P.O. Box 365, Pietermaritzburg.
89. Pilkington Group European Medical Aid Society, P.O. Box 111, Springs.
90. Polliack Group Medical Aid Society, P.O. Box 3008, Johannesburg.
91. Pongola Sugar Milling Co. Ltd. Medical Benefit Fund, P.O. Box 194, Durban.
92. Post Office Medical Aid Society, P.O. Box 303, Germiston.
93. Pretoria Municipal Employees' Sick Fund, P.O. Box 408, Pretoria.
94. Pretoria News Medical Benefit Society, P.O. Box 439, Pretoria.
95. Pretoria Portland Cement Co. Ltd. No. 1 Works (Herlaer Place, C.P. (Only certain groups as indicated on p. 1064
96. Pretoria Portland Cement Co. Ltd. No. 2 Works Medical Benefit Society, P.O. Box 7, Slurry, Western Transvaal.
97. Pretoria Portland Cement Co. Ltd. No. 3 Works (Jupiter) Medical Aid Society, P.O. Box 73, Cleveland, Transvaal.
98. Pretoria Portland Cement Co. Ltd. No. 4 Works Medical Aid Society, P.O. Box 26, Orkney, district Klerksdorp.
99. Printing Industry Medical Aid Society, P.O. Box 1993, Pretoria.
100. Prudential Medical Aid Scheme, P.O. Box 1097, Johannesburg.
101. Rand Water Board Sick Fund, P.O. Box 1127, Johannesburg.
102. Randles Bros. & Hudson Ltd. (Durban) Sick Benefit Fund, P.O. Box 1046, Durban.
103. Randles Bros. & Hudson Ltd. (Johannesburg) Employees' Sick Benefit Fund, P.O. Box 2678, Johannesburg.
104. 'Rennie' and 'The Consolidated' Employees' Medical Aid Fund, P.O. Box 1006, Durban.
105. Reynolds Bros. Ltd. Medical Benefits Fund, 301 Smith Street, Durban.
106. E. S. & A. Robinson (Pty.) Ltd. Medical Aid Society, P.O. Box 293, Germiston.
107. Royal-Globe Medical Aid Fund, P.O. Box 83, Cape Town.
108. Safim Medical Aid Society, P.O. Box 233, Vereeniging.
109. Safmarine Medical Aid Society, P.O. Box 2171, Cape Town.
110. Safnit Mills Medical Aid Fund, P.O. Box 11, Jeppestown, Johannesburg.
111. SANLAM Siektefonds (alle takke), Posbus 1, Sanlamhof, K.P.
112. SANTAM Onderlinge Mediese Hulpvereniging, Posbus 653, Kaapstad.
113. Shell Medical Aid Society (S.A.), P.O. Box 2231, Cape Town.
114. S.A. Breweries Medical Aid Society, P.O. Box 1099, Johannesburg.
115. S.A.K.A.V. Sick Benefit Fund, P.O. Box 33, Paarl.
116. S.A. Mutual Fire & General Insurance Co. Ltd. Staff Medical Aid Fund, P.O. Box 516, Johannesburg.
117. S.A. Mutual Life Assurance Society Staff Medical Aid Fund, P.O. Box 66, Cape Town.
118. S.A. Mutual Medical Aid Society, P.O. Box 90, Howard Place, C.P. (Only certain groups as indicated on p. 1064 of this issue of the *Journal*.)
119. S.A. National Sickness & Accident Insurance Co. Ltd. (SANSOM), Box 25, Sanlamhof, C.P. (Only certain groups as indicated in a supplement to the *Journal* of 26 November.)
120. S.A. Press Association Medical Aid Society, P.O. Box 7766, Johannesburg.
121. S.A. Pulp & Paper Industries Medical Benefit Fund, Tugela Mill, P.O. Mandini, Zululand.
122. S.A. Sugar Association Medical Benefits Fund, P.O. Box 2160, Durban.
123. S.A. Teachers' Association Medical Aid Society, 12 Bellevue Road, Sea Point, C.P.
124. S.A. Torbanite (Boksburg) Medical Aid Society, P.O. Box 5083, Boksburg North.
125. South Atlantic Corporation Medical Aid Society, P.O. Box 1628, Cape Town.
126. Southern Medical Aid Society, P.O. Box 42, Cape Town.
127. Springs Industrial Benefit Society, P.O. Box 554, Springs.
128. Standard Brass Medical Aid Society, P.O. Box 229, Benoni.
129. Steeldale and Union Joinery Medical Aid Society, P.O. Box 1210, Johannesburg.
130. Sun Insurance Office Ltd. Staff Medical Aid Fund, P.O. Box 429, Johannesburg.
131. Sydmore Sick Benefit Society, P.O. Box 8851, Johannesburg.
132. Syfrets' Medical Aid Society, 24 Wale Street, Cape Town.
133. Traduna Medical Aid Fund, P.O. Box 8791, Johannesburg.
134. Transvaal Corundum Associated Asbestos Medical Aid Society, P.O. Box 72, Pietersburg, Transvaal.
135. Transvaal Society of Accountants Medical Aid Fund, P.O. Box 2995, Johannesburg.
136. U.L.A. Medical Aid Society, P.O. Box 4589, Johannesburg.
137. Umzimkulu Sugar Co. Ltd. Medical Aid Fund, P.O. Box 43, Durban.
138. United Banks' Medical Aid Society, P.O. Box 1242, Cape Town.
139. United Building Society Medical Aid Fund, P.O. Box 7735, Johannesburg.
140. University of the Witwatersrand (Johannesburg) Staff Medical Aid Fund, Milner Park, Johannesburg.
141. Village Board of Management of Welkom Medical Aid Society, P.O. Box 708, Welkom, O.F.S.
142. Wright, Boag & Head, Wrightson Sick Benefit Fund, P.O. Box 183, Benoni.
143. Yorkshire Medical Aid Society, P.O. Box 2755, Johannesburg.

MEDIESE BYSTANDSVERENIGINGS WAT VRY KEUSE VAN DOKTER ALLEEN VIR SPESIALISTEDIENSTE TOELAAT

MEDICAL BENEFIT SOCIETIES WHICH ALLOW FREE CHOICE OF DOCTOR FOR SPECIALIST SERVICES ONLY

1. Begbie Medical Benefit Fund, P.O. Box 192, Middelburg, Transvaal.
2. Brakpan Power Station Sick Benefit Society, P.O. Box 1, Brakpan.
3. Breyten Coalfields Benefit Society, P.O. Box 6, Estantia, Transvaal.
4. Broken Hill Mine Employees' Medical Specialist Fund, P.O. Box 45, Broken Hill.
5. De Beers Consolidated Mines Limited Benefit Society, P.O. Box 616, Kimberley.
6. Durban Roodepoort Deep Ltd. Benefit Society, P.O. Box 193, Roodepoort.
7. Jagersfontein Mine Benefit Society, P.O. Box 2, Jagersfontein, O.V.S.
8. Krugersdorp Municipal Employees' Medical Benefit Society, P.O. Box 101, Krugersdorp.
9. Northern Rhodesia Mine Employees' Medical Specialist Fund, P.O. Box 134, Kitwe, N.R.
10. Public Utility Transport Corporation Sick Fund, P.O. Box 9571, Johannesburg.
11. Randfontein Estates Employees' Sick Benefit Society, P.O. Box 37, Randfontein.
12. Roodepoort-Maraisburg Municipal Employees' Sick Benefit Society, P.O. Box 217, Roodepoort.
13. Roodepoort-Maraisburg Non-Scheduled Mines, and Industries' Benefit Society, P.O. Box 225, Roodepoort.
14. Rosherville-Maraisburg Benefit Society, P.O. Box 99, Cleveland, Johannesburg.
15. Sasol Medical Benefit Society, P.O. Box 80, Sasolburg.
16. Simmer Pan Medical Society, P.O. Box 103, Germiston.
17. Springs Mines Benefit Society, P.O. Box 54, Springs.
18. Tongaat Sugar Company Medical Benefit Scheme, P.O. Box 5, Maidstone, Natal.
19. Transvaal Jewellers' & Goldsmiths' Sick Benefit Fund, P.O. Box 8530, Johannesburg.
20. Tweefontein Colliery Employees' Benefit Society, Tweefontein Colliery, P.O. Coalville, Transvaal.
21. Western Province Building & Allied Trades Sick Fund, P.O. Box 2013, Cape Town.
22. Witbank Coalfields Benefit Society, P.O. Box 26, Witbank.
23. Witbank Power Station Medical Benefit Society, P.O. Box 197, Witbank.

SOUTH AFRICAN MUTUAL MEDICAL AID SOCIETY

Appended hereto is a list of groups which conform to the requirements of the Medical Association for approved medical aid societies. These groups received the approval of Federal Council at the meeting held in Vereeniging on 19 - 21 October.

1. Adami's Mining & Electrical Supplies (Pty.) Ltd., Welkom.
2. Adolph Mosenthal & Co. Ltd., Port Elizabeth.
3. Adriennes, Port Elizabeth.
4. A. & T. Outfitters, Bloemfontein.
5. Bäckerei & Konditorei (P. Thiemann), Otjiwarongo.
6. Balatum S.A. (Pty.) Ltd., Durban.
7. I. Barbakow, Bloemfontein.
8. Bate, Chubb & Dickson, East London.
9. E. Baumgart (Pty.) Ltd., Walvis Bay.
10. Behrendt & Kronheim, Cape Town.
11. Betterman's, Bloemfontein.
12. Bontiesigns, Bloemfontein.
13. K. Borowski Furriers, including Amsterdam Furriers (Pty.) Ltd., Johannesburg.
14. Brockmann & Kriess (Pty.) Ltd., Windhoek.
15. Bülbring Bros. (Pty.) Ltd., Port Elizabeth.
16. Cape Manuf. Engineers (Pty.) Ltd., Cape Town.
17. Central Shoe & Repair Works, Bloemfontein.
18. Ceramic Builders Suppliers (Pty.) Ltd., Claremont.
19. Cohen's Bottle Store, Port Elizabeth.
20. Crawford McKenzie & Co., East London.
21. Croft, Magill & Watson, Kimberley.
22. T. W. Dersley, Port Elizabeth.
23. E. C. de Witt & Co. (S.A.) Ltd., Port Elizabeth.
24. Downing & Attwood (Pty.) Ltd., Port Elizabeth.
25. Dunbar & Kenley, Witbank.
26. Eastern Province Engineers (Pty.) Ltd., Port Elizabeth.
27. Fr. Eberlanz, Luderitz.
28. Ernst Holtz (Pty.) Ltd., Windhoek.
29. Ferreira Bros., Port Elizabeth.
30. Fletcher & Cartwrights Ltd., Cape Town.
31. Giminghams Garage, King William's Town.
32. C. L. Gregory, Port Elizabeth.
33. Apie Griesel & Co., Kroonstad.
34. B. Grundleger (Pty.) Ltd., Windhoek.
35. Handy House, Port Elizabeth.
36. D. J. Hattingh Co. (Pty.) Ltd., Windhoek.
37. Heinz J. Betz & Co., Port Elizabeth.
38. A. E. Howes (Pty.) Ltd., East London.
39. Improved Sheetmetal Manufacturers, Bloemfontein.
40. Isaacs Bros. & Co. (Pty.) Ltd., Port Elizabeth.
41. Jewellery Trade Co. (Pty.) Ltd., Johannesburg.
42. Johannesburg Jewellers, Kroonstad.
43. R. Kaplan, Bloemfontein.
44. Kapps-Hotel (K. Ohlhof), Luderitz.
45. The Kelt Engineering Co. (Pty.) Ltd., Kroonstad.
50. C. Kennedy Humphrey & Son, East London.
51. King Furnishers (Pty.) Ltd., King William's Town.
52. Kohler Bros. (Timber) Ltd., Port Elizabeth.
53. Kroonstad Motors (Pty.) Ltd., Kroonstad.
54. Küstner's Garage, Otjiwarongo.
55. Eric Lamb, Bloemfontein.
56. Le Roux & Co., Port Elizabeth.
57. L. S. Luck, East London.
58. Marble, Lime & Ass. Ind. Ltd., Johannesburg.
59. A.D.B. McGregor & Co. (Pty.) Ltd., Port Elizabeth.
60. Francis Meaker, Somerset East.
61. Frank Meaker, Somerset East.
62. H. Meyer, Luderitz.
63. Michel Louis & Sons (Pty.) Ltd., Bloemfontein.
64. Modehaus 'Gertrude', Luderitz.
65. Model Slaghuis, Theunissen.
66. Mooi River Supply Store (Pty.) Ltd., Durban.
67. Morgan, Steenkamp & Schwims (Pty.) Ltd., Welkom.
68. Msenge Ridge Dairy Farm, East London.
69. Norris Garage (Pty.) Ltd., East London.
70. John Orr & Co., Bloemfontein.
71. Ottilie Nitzsche-Joseph-Reiter, Windhoek.
72. Otjiwarongo Sau-Masch Schlosserei, Otjiwarongo.
73. Otto Mühr (Pty.) Ltd., Windhoek.
74. George M. Parker (Pty.) Ltd., Port Elizabeth.
75. K. N. Paterson & Co., East London.
76. Percys, Bloemfontein.
77. H. Pinshaw & Sons, Bloemfontein.
78. Pyott Ltd., Johannesburg.
79. Radio-Service Walvis Bay (Pty.) Ltd., Walvis Bay.
80. Regal Trading Co. (Pty.) Ltd., Johannesburg.
81. John Roderick & Neill Ltd., Bloemfontein.
82. Serck Radiators (S.A.) (Pty.) Ltd., Cape Town.
83. S.A. Scottish Finance Corporation Ltd., Johannesburg.
84. Shapiros Wholesale Distr. (Pty.) Ltd., Bloemfontein.
85. S.W.A. Electrical Engineers (A. Hegewisch), Otjiwarongo.
86. S.W.A. Fishing Industries Ltd., Luderitz.
87. G. A. Smith (Pty.) Ltd., Port Elizabeth.
88. Strachan & Dowling, Durban.
89. Syfret, Godlonton & Low, Cape Town.

90. T. C. M. Plumbing (Pty.) Ltd., Bloemfontein.
91. Ted Hamer General Dealer, Theunissen.
92. Theunissen Hotel, Theunissen.
93. Transafrican Radio Co., Bloemfontein.
94. Troskie & Botha, Port Elizabeth.
95. J. H. van Heerden, Theunissen.
96. Van Riebeeck Dry Cleaners, Port Elizabeth.
97. Van Rynevelds (Pty.) Ltd., Kroonstad.
98. Voxan (Pty.) Ltd., Port Elizabeth.
99. Warenhaus Dreybrodt, Otjiwarongo.
100. Waverley Café, Bloemfontein.
101. Wecke & Voigts, Okahandja.
102. T. Wilcox & Co. (Pty.) Ltd., East London.
103. Wilson Engineering Works, Port Elizabeth.
104. Windy Hill Wattle Co. Ltd., Durban.
105. Xmeco (Pty.) Ltd., Port Elizabeth.
106. Eduard Zimmer (Pty.) Ltd., Windhoek.

UNIVERSITEITSNUUS : UNIVERSITY NEWS

UNIVERSITY OF THE WITWATERSRAND EXAMINATION RESULTS

The following candidates have completed all the requirements of the Sixth Professional Examination for the degree of M.B., B. Ch.:

Agrotis, E. N.
Auerbach, R.
Bengani, D. D.
Berger, G. M. B.
Boner, G.
Bosman, C. K.
Botha, P. A.
Cochrane, R. I.
Cohen, M.
Crosley, A. I.
Davis, G.
Dawes, M. E.
Dimopoulos, P.
Dove, E.
Edge, K. R.
Fanaroff, A. A.
Feldman, J. D.
Geldenhuys, A. R.
Glazer, H.
Gottlieb, A. M.
Hall, L. U.
Heyns, A. du P.
Hollis, R. R.
Howell, M. E. O.
Hurwitz, M. B.
Jackson, R. A.
Janse van Rensburg, M. P.
Jassat, E.
Kahn, L. (2nd Class Honours)
Kahn, L. B.
Kalell, A.
Katz, G.
Kretzmar, T. D.
Kuming, B. S.
Kussel, J. J.
Lampert, G.
Landsberg, P. G.
Lee, J. A.
Lissoos, I.
Livni, N. M.
Maass, G. F. A.
Mauff, A. C.
McCusker, V. I.
Meyers, A. M.
Milunsky, A.
Nissenbaum, M. M.
Otten, R. B.
Ou Tim, S.
Palmer, R. I.
Parry, D. G.
Paton, D. F.
Plit, M.
Prentice, B. R.
Pretorius, J.
Ravdel, A.
Rosenberg, C. M.
Rossouw, D. P.
Rubenstein, A. H. (1st Class Honours)
Schneider, G.
Shapiro, I.
Shulman, G.
Silver, H.
Sims, F. H.
Stuart, J. L. F.
Urbani, P.
van As, A. W. W.
van Veen, E. J. W. B.
Vinik, A. I.
Willies, L. J. S.
Wilson, W.
Zail, S. S. (2nd Class Honours)
Zeilinga, J. A.

WORLD LIST OF FUTURE INTERNATIONAL MEETINGS

ALTERATIONS AND ADDITIONS NOTIFIED DURING NOVEMBER 1960

Commission for Technical Cooperation in Africa South of the Sahara, 16th Session, Lagos, Nigeria, February 1961. Pvt. Mail Bag 2359, Lagos, Nigeria.

European Seminar on Water Pollution Control, Geneva, late February 1961. Economic Commission for Europe, United Nations, Palais des Nations, Geneva, Switzerland. (With WHO, FAO and IAEA.)

Pan American Medical Association, Congress, February 1961. 745 Fifth Avenue, New York, N.Y., USA.

Symposium on Public Nutrition Problems (Symposium über Fragen der Volksernährung), Berlin, March or May 1961. German Academy of Sciences, Otto Nuschke Str. 22 - 23, Berlin W.8, Germany.

International College of Podology, Congress of Anatomy, Physiology and Pathology of the Heel, Monaco, 28 - 30 April 1961.

French-language Association of Scientific Psychology (Association de Psychologie Scientifique de Langue Française), 7th Session, Amsterdam, 1st week April 1961. Prof. Paul Fraisse, Secrétaire Général, 28 rue Serpente, Paris 6e, France.

Psychotherapy Week, 11th, Lindau, Germany, 1 - 6 May 1961. Secretariat, Dienerstrasse 17, Munich 2, Germany.

Middle East Medical Assembly, 11th, Beirut, 5 - 7 May 1961. Dr. Raif Nassif, Chairman of the Assembly, American University, Beirut, Lebanon.

Federation of Societies of French-speaking Gynaecologists and Obstetricians (Fédération des Sociétés de Gynécologie et d'Obstétrique de Langue Française), 19th Congress, Nancy, 29 - 31 May 1961. Prof. Marcel Ribon, Secretary General, 1 pl. de la Commanderie, Nancy, France.

International Association for Bronchology, 11th Congress, Rome, ? May 1961. Prof. Montanini, Instituto Carlo Torlanini, Rome, Italy.

International Organization against Trachoma, Meeting, Paris, May 1961. Dr. Jean Sédan, Secretary General, 94 rue Sylvabelle, Marseilles, France. In connection with the 68th Congress of the French Society of Ophthalmology. The League against Trachoma is also meeting.

Tenth International Congress of Kinesitherapy and Physical Re-education, Liège, 21 - 25 June 1961. Emmanuel Crenier, Secretary General, 3 avenue Malvoz, Liège, Belgium.

IN DIE VERBYGAAN : PASSING EVENTS

Mr. J. M. Edelstein, of Johannesburg, has recently returned from an extended tour of the USA and Great Britain. At the Annual Meeting of the British Orthopaedic Association an Honorary Fellowship was conferred on Mr. Edelstein.

* * *

Dr. and Mrs. J. J. Jacobson, of Cape Town, have recently returned from an overseas visit. Dr. Jacobson has resumed his practice at 17 Church Square after his postgraduate study vacation.

Drs. Max P. Shapiro and A. J. H. Henning, radiotherapists, have changed their address to 'Princess', 69 Esselen Street, Hillbrow, Johannesburg, telephone 44-6256. This number does not appear in the current Telephone Directory.

* * *

Drs. Max P. Shapiro en A. J. H. Henning, radioterapeute, het hulle adres verander na 'Princess', Esselenstraat 69, Hillbrow, Johannesburg, telefoon 44-6256. Hierdie telefoonnommer verskyn nie in die jongste Telefoongids nie.

IN MEMORIAM

JOSEPH REUBEN EIDELMAN, M.B., B.CH. (RAND)

Dr. A. L. Agranat, of Johannesburg, writes:

Dr. Joseph R. Eidelman died on 13 November 1960. I have known him for the past 14 years and have been fortunate to be in a position to appreciate his fine qualities and his excellent character in both a personal and a professional capacity.

Dr. Eidelman

He was born in 1901 and came to South Africa when he was 8 years old. At that time he lived in Johannesburg where he matriculated and then spent several years in the civil service and doing other work in order to earn enough to pay for his medical course. He qualified M.B., B.Ch. at the University of the Witwatersrand in 1927, and served a period of housemanship at the Johannesburg Hospital under the late Mr. Joseph Levin. He started practice at Krugersdorp and later settled at Randfontein where he was in active practice for about 30 years.

Dr. Eidelman succeeded Dr. P. D. Hamilton as Senior Medical Officer to the Randfontein Estates Benefit Society, in which capacity he served at the Robinson Hospital for about 25 years. As Chief of the Hospital he was held in the highest esteem by his colleagues, the nursing staff and his patients, all of whom were devoted to him. In appreciation of his services to the hospital, a theatre was named after him when he resigned about 5 years ago for reasons of health.

His ability as a doctor was outstanding; he was an able surgeon and had a wide knowledge of general medicine. He was in the front rank of practising doctors and kept abreast of modern developments in medicine by adequate reading.

For 10 years he met the trials of ill health with tremendous fortitude and courage. His concern was always about others and his sincerity and pleasant personality set an example to many. Those of us who knew the extent of his physical sufferings were amazed at his determined avoidance of any reference to the subject.

He also took a keen interest in affairs outside medicine. He was a Rotarian for many years and, at the time of his death, was the President of the Randfontein Mines Bowling Club. He was a man who was deeply devoted to his family and he had hoped to see his only son, John, qualify before the inevitable occurred. It is a sad thought that he was denied this pleasure because his illness caused John to postpone his final examinations.

The very large gathering at his funeral bore testimony to his popularity and to the affection of his many friends. He will be sadly missed by everybody who knew him. The sincerest sympathy is extended to his wife and children.

LOUIS MIRVISH, M.A., M.B., CH.B. (CAPE TOWN), M.R.C.P. (LOND.)

Drs. Louis Herrman and Arthur Landau, of Cape Town, write:

The death of one of South Africa's leading physicians, Dr. Louis Mirvish, at the age of 62, occurred on 18 November to the deep regret of a wide circle of relatives, friends and colleagues, students and patients. Dr. Mirvish was senior physician at the Groote Schuur Hospital and a part-time lecturer in Medicine at the University of Cape Town.

Dr. Mirvish

A son of the late learned and much-respected Rabbi of the Orthodox Hebrew Congregation, he inherited the best traditions of the ancient Jewish love of learning. He came to South Africa with his parents as a small child and attended the old Hopemill and Normal College schools before proceeding to the University. It was at the University of Cape Town that he gained his M.A. in 1918, and graduated in medicine and surgery in 1922, one of the 2 first graduates of the then new Medical School. After some years as a general practitioner and lecturer in physiology in the University, where he interested himself in biological research, he went to Europe where he did postgraduate study in London, Berlin and Vienna. Returning to Cape Town in 1932 he established practice as a specialist in gastro-enterology and metabolic diseases, in which capacity his ability was such that he came in time to enjoy the widespread confidence and respect of his medical colleagues in the Union of South Africa and even beyond its borders.

He was a man of wide culture and enquiring mind with intellectual interests that embraced not only an enthusiasm for the advance of scientific knowledge, but many other aspects of culture, more especially art, history and antiquities. He dearly loved intellectual intercourse with a few chosen friends where he talked with ease and knowledge, contributing many an original idea to the discussion. In his enthusiasm for art he encouraged and assisted painters and sculptors, and his love of works of antiquarian value and *objets d'art* led him to make an interesting little collection as well as to work for the establishment, over a period of several years, and finally to achieve success in the founding, of the Jewish Museum in Cape Town.

He wrote and published papers on biological subjects, the results of his researches, and contributed to the medical press on gastro-enterology and the treatment of diabetes. He compiled the Diet Book for the Groote Schuur Hospital and was engaged at the time of his death in writing a more extensive work on the same subject. In London, about 1930, he collaborated with a colleague in writing a book on the stomach. But although his part of the work was completed before he returned home, his name was omitted from the title page. It is characteristic of his tolerant disposition that he suffered the slight in silence.

A broad humanity informed his life and work. His 'cases' were persons and fellow-creatures, and their hopes and fears were his concern no less than their physical symptoms. The unfortunate commanded his sympathy, and he assisted the partially disabled by founding the Sheltered Employment Society to aid them to work, to dignity and independence. Understanding lighted his path and in his heart was kindness.

Mr. George Sacks, of Cape Town, writes:

I first met Louis Mirvish in the biochemistry class in a small room at the top of Government Avenue. He was a demonstrator working for his M.A. degree. Years later we worked togther in the Physiology Department in the new buildings which had gone up in Observatory. Physiology was, I think, his first love and when he became a physician it was always some physiological problem which excited him most.

He had a great capacity for making friends outside medicine. It was in his house that one would meet stormy petrels like Lancelot Hogben, then winging his way through Cape Town university life like a meteor, Sewell, Zuckerman, and artists

of all kinds and of varying merit. The art of conversation flourished in his sitting-room.

His practice was unique. A pioneer in gastro-enterology, he drew his patients from all over Africa. His appointment book would be headed 'Belgian Congo month', 'Rhodesia month', Johannesburg' and so on. They came in droves to get a sympathetic hearing, a thorough examination, skilful and kindly advice.

At Groote Schuur Hospital where once more we were colleagues he was a pillar of strength, a stern critic when occasion arose and a loyal friend. He was the gentlest of men, entirely uninterested in the financial side of practice (he must have had the largest *pro Deo* practice in Cape Town) and his greatest interest lay in his ward patients.

We miss him more than words can tell.

ANDREW CAMPBELL WATT, M.B., CH.B., M.R.C.P. (EDIN.)

Prof. Norman M. Dott, Head of the Department of Neurosurgery, Edinburgh University, writes:

The tragic and untimely death of Dr. Andrew Campbell Watt, of Johannesburg, focuses attention on British wartime debt to those young doctors of the Commonwealth who threw all they had into service with her medical profession during the stress of war. Dr. Watt's father received his medical education in Edinburgh and became an important figure in medical circles in Johannesburg. He sent his son back to the 'old school' where he graduated in 1938.

After holding posts in psychiatry and casualty, the outbreak of war in September 1939 found Andrew Watt house surgeon in the Department of Surgical Neurology of the Royal Infirmary, Edinburgh. A few months later the Department was moved, for strategic reasons, to Bangour Hospital near Edinburgh, and Watt moved with it — indeed one might more properly say he moved it! In those days all available young British doctors were being rapidly recruited for active military service. Dr. Watt dealt with a most formidable task as resident surgeon in the Edinburgh Brain Injuries Unit established at Bangour. He carried a load — about 900 admissions of major neurological injuries and disease annually and 350 major neurosurgical operations — as single-handed resident surgeon, that would have required at least 4 residents in peace-time. He displayed extraordinary stamina, energy, and ability in organizing the emergency unit — about half civilian and half armed-forces cases. His loyalty to the patients, his colleagues, and to the high standards of practice he had acquired, was magnificent. During the war we were able, not only to maintain standards, but also to improve on them — developing cerebral angiography, treatment of aneurysms and epilepsy, etc. and greatly improving the management of head injuries and rehabilitation measures. That surgical neurology in Edinburgh emerged from the war with a larger potential for good and with improved standards of practice, was due to Watt's magnificent work during those most difficult years 1940 - 1943. I estimate that he averaged an 18 hours' working day and night in that period.

Having achieved this, and the Unit having become stabilized under war conditions, Andrew Watt felt the call to personal military service at the active fronts. He joined the RAMC and with his special experience was posted to the principal Neurological Military Hospital in England, near Oxford. There he served with the principal neurologists and neurosurgeons — Brigadier Generals Hugh Cairns and George Riddoch, and Air Vice-Marshal Charles Symonds. He was posted as neurologist to a mobile team with the Normandy invasion forces and acquired fame for his extraordinary capacity for sustained effort at high standards. He could do 3 men's work and do it better than 3 men! After the Armistice he was stationed in Belgium for a considerable period, where he came under the influence and gained the friendship of Ludo van Bogaert, the great neurological figure at Antwerp.

Dr. Watt stayed with us in Edinburgh for 2 years after the Armistice helping to clear up the neurological aftermath of war and finding time to pass the M.R.C.P.E. in 1947.

His subsequent career as neurologist in Johannesburg again required much of his enthusiasm and pioneering spirit, since surgical neurology was developing there and required his strong support, which he freely gave. He had attained a position of great influence, importance, and respect and, at the age of 46, his potential for good was enormous.

At this point, and in the course of responding to an emergency call, came his untimely end. The loss of this able and experienced specialist is a grievous one to his colleagues and his profession. The loss to those many of us in Britain who were deeply in his debt, who knew his strong friendship, his quiet humour, his grasp of medical and human affairs, his high ethical standards, and his selfless devotion to his patients, is truly tragic. Our deepest sympathy goes out to Mrs. Watt and to his young daughter and son. We are grateful for the memory of such a loyal comrade, and we are devastated by his untimely death.

NUWE PREPARATE EN TOESTELLE : NEW PREPARATIONS AND APPLIANCES

VANQUIN SUSPENSION

Parke, Davis Laboratories (Pty.) Ltd., announce the introduction of Vanquin Suspension, a single-dose treatment for threadworm infestation, and supply the following information:

It has been estimated that more than 800 million people, over one third of the earth's population, are hosts to various types of helminths. Threadworms are perhaps one of the most prevalent parasitic infestations, especially in children. The incidence of threadworm infestation throughout the world was estimated by Stoll in 1947 to be more than 200 million and there is little doubt that the great increase in world population since 1947 has been accompanied by an increase in the number of threadworm infestations.

Vanquin Suspension, is a new and unusually effective single-dose treatment for threadworm. Clinical reports have shown Vanquin Suspension to achieve nearly 100% cure rate.

Description. Vanquin Suspension (pyrvinium pamoate, Parke-Davis) is a new medical agent having as its active ingredient pyrvinium pamoate, a deep-red sparingly-soluble salt of a cyanine dye.

Indications. Vanquin Suspension has been shown to be singularly effective against threadworm. In a single dose Vanquin Suspension will clear the majority of infestations. It is truly vermicidal and is the ideal means of breaking the threadworm life-cycle. The simplicity of the treatment makes Vanquin Suspension particulary suitable for eradication programmes in households or institutions.

Dosage and administration. Orally, in a single dose equivalent to 1 5 ml.-teaspoonful of Suspension per 22 lb. (10 kg.) bodyweight approximately. Parents and patients should be told that Vanquin will colour the stools a bright red and if spilled will stain.

Side-effects. In general Vanquin is well tolerated and in the recommended doses there have been few side-effects. A few patients have experienced nausea or vomiting. Mostly these were older children or adults who received relatively large doses. In several cases it is doubtful whether the gastric upset was drug-induced. Toxicity due to overdosage is unlikely as Vanquin is not appreciably absorbed.

Packing. Vanquin is a strawberry-flavoured suspension containing the equivalent of 10 mg. pyrvinium base per ml. in bottles of 30 ml.

Further information may be obtained from Parke, Davis Laboratories (Pty.) Ltd., P.O. Box 24, Isando, Transvaal.

BRIEWERUBRIEK : CORRESPONDENCE

TREATMENT OF PLANTAR WARTS WITH ULTRASONIC THERAPY

To the Editor: The satisfactory treatment of plantar warts has baffled the profession for many years. The surgeon excised them or scraped them, deep-X-ray therapy was administered by the radiotherapist, and the physical medicine specialist performed a diathermy cauterization; but the results of treatment were not uniformly successful and, besides the period of disability during treatment, there were frequent recurrences.

During the past year I have treated a number of cases with ultrasonic therapy, and the results have been exceptionally good. Each wart is treated once a week at an intensity of 0·6 watts per square cm., for 15 minutes. Generally, not more than 10 treatments are necessary.

I should like my colleagues to try this method of treatment and, if possible, publish their results.

H. D. Epstein

225 Central House
Central Street
Pretoria
24 November 1960

URETERIC INJURIES

To the Editor: The Editorial[1] and Mr. Jacobson's article 'Ureteric injuries in gynaecology'[2] are timely reminders that there is much room for improvement in prevention, diagnosis, and treatment of ureteric injuries.

Textbook information on this subject reflects the practical deficiencies in experience of its authors as do the majority of publications on this subject, so that pooling of knowledge and experience — such as that obtained by Mr. Jacobson from his series of 3 cases — is valuable. My own special interest in this problem prompts me to make comments, limited by the space available for correspondence, and based upon a series of 23 such cases referred to me from various parts of Natal during the past 6 years.

Firstly, I would suggest that a more suitable title for Mr. Jacobson's article would have been, 'Ureteric injuries in pelvic surgery' rather than 'in gynaecology' for whilst I am critical of the practical training programme given to specialist gynaecologists in its tendency to produce mere efficient 'midwife/hysterectomists' my experience prompts me to suggest that the majority of ureteric injuries occur at the hands of those outside our speciality. Gynaecologists, for instance, only undertake a minority of the pelvic surgery among non-Europeans, who constitute the bulk of our population, yet the more formidable surgical problems presented by this section of the community render them especially vulnerable to ureteric injury.

Arising out of this fact, it is interesting to note that the conventional aetiological factors emphasized by Mr. Jacobson are not the most important in many areas of South Africa. For instance, 4 out of 23 of the ureteric injuries in my series resulted from Caesarean sections and a further 5 from blind repair of vesico-vaginal fistulae. The danger of ureteric injury especially when suturing a thinned lower segment on the left side, which has been drawn up or torn down towards the vagina or into the left uterine artery, is not sufficiently appreciated; nor are the precise ureteric relationships defined with sufficient care in the repair of many vesico-vaginal fistulae. In the latter regard I am loath to leave the subject of vesico-vaginal fistulae without seizing upon the opportunity to deplore the frequency with which uretero-colic anastomoses (after failure to close vesico-vaginal fistulae) continue to be performed by individuals who are unprepared to refer cases to centres where reconstructive surgery offers every prospect of cure. I have seen no less than 5 such cases over the past 2 years — 3 in renal failure.

I should like to congratulate Mr. Jacobson on his active and aggressive attitude towards the early repair of ureteric injuries and his conservative approach towards nephrectomy.

Contrary to current urological practice I have adopted this aggressive approach towards the early repair of ureteric fistulae as the only logical course since 1955, and have had to perform only 2 nephrectomies (with a third performed at Edendale) during the follow-up of 23 ureteric fistulae. This contrasts markedly with the all too prevalent rapid recourse to nephrectomy in these cases.

I believe that the early repair of ureteric fistulae after Wertheim's hysterectomy is particularly important. In my hands an intraperitoneal approach to repair has proved more advantageous than the extraperitoneal approach suggested by Mr. Jacobson, because it offers better opportunities to study the relationships resulting from the previous operation, renders mobilization of tissues easier, and affords more flexibility in the selection of that technique which will permit of an accurate anastomosis without any semblance of tension.

Finally, in regard to prevention, we are indebted to the *Journal* and Mr. Jacobson for much sage advice, but you will agree that such admonitions are mere repetition of similar statements which have appeared in standard textbooks and publications over a great many years. Your approach is exactly the same as that of the general surgeons 50 years ago when they told obstetricians, who did not have the training to undertake abdominal surgery, that they should really practise a better standard of obstetrics and take more precautions to avoid unnecessary Caesarean sections!

No real improvement has resulted and none obviously will result until departments of gynaecology provide a sound training programme of pelvic surgery inclusive of uro-gynaecology instead of the prevalent restricted approach of producing 'genital tractologists' (with blinkers on). The problem is not solved by sending a house surgeon or registrar to a urology department for 6 months, for he returns with a very incomplete practical knowledge of the problem as it faces the surgeon undertaking pelvic surgery — as I know from personal experience of such individuals. A sound training can only be afforded if uro-gynaecology is developed as part of pelvic surgery within a gynaecology department. Those urologists who are especially interested in the important overlap of our specialities could well be assimilated for such a programme with mutual benefit, for there is no doubt that many gynaecological misadventures of our urological colleagues — unheralded by revealing moisture — pass unsung, although not unknown, to their gynaecological colleagues.

This has been the training afforded to gynaecologists in the Durban Medical School since 1956 where urethro-cystoscopic procedures are second only in frequency to diagnostic curettages in providing the trainee with an appreciation of the importance of the lower urinary tract in gynaecology, and in producing an individual who has a sound practical knowledge of endoscopic procedures. Their confidence in handling the ureters is likewise enhanced by their obligation to display the pelvic course of the ureter before proceeding with a hysterectomy in each case, and I firmly believe that the extra few minutes spent in this dissection is in the interests of the patient's safety, (although at the sacrifice of a little additional oozing), a few minutes of extra time, and some loss of the spectacular rapid, dry surgery which can be attained by grasping and stitching large blind pedicles.

Derk Crichton, D.Phil., F.R.C.S., F.R.C.O.G.
Professor of Gynaecology and Obstetrics

University of Natal
Durban
24 November 1960

1. Editorial (1960): S. Afr. Med. J., **34**, 907 (22 October).
2. Jacobson, I. (1960): *Ibid.*, **34**, 901.